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ALIEN PHYSICIANS TRAINING IN HOSPITALS IN THE UNITED STATES

Harold S. Diehl, M.D., Minneapolis, Edwin L. Crosby, M.D., Chicago
and
Paul K. Kaetzel, B.A., Washington, D. C.

In the past few years there has been a great increase in the number of alien physicians on house staffs of hospitals in the United States. Temporary immigration of alien physicians for the purpose of taking training has been greatly facilitated by the U. S. Information and Educational Exchange Act of 1948, which became effective in July, 1949. Since that time hospitals have been authorized to offer internships and residencies to alien physicians after securing approval of their educational programs by the Department of State. Approvals have now been granted to about 800 hospitals.

Yearly surveys of house staffs in training at civilian hospitals approved for internships and/or residencies have been conducted by the Health Resources Advisory Committee of the Office of Defense Mobilization. These surveys have brought together information concerning alien physicians serving as interns and residents. The report that follows is based on surveys conducted for the 1953-1954 training year. Coverage represented 99% of all interns and residents in training at civilian hospitals approved for such training. The term "civilian hospitals" includes all hospitals except those operated by the Department of Defense and the Public Health Service.

SURVEY FINDINGS

A total of 5,589 alien physicians held appointments as interns, residents, or fellows on house staffs at civilian hospitals approved for training in July, 1953, the beginning of the 1953-1954 training year. Three years earlier the total was 2,072. Of the July, 1953, total, 1,787, or about one-third, were first year interns and the remaining two-thirds, 3,802, were residents or fellows. Alien physicians represented 22% of the total house staffs at approved civilian hospitals (table 1).

Without the services of aliens hospitals would have a considerably greater proportion of unfilled positions than the present 20% for residencies and 30% for internships. Many young physicians who would normally be taking postgraduate medical education are on active military duty.

For the most part, aliens were found to be receiving training in general hospitals, chiefly those without major teaching affiliations. Over 60% of all alien interns and residents were in nonteaching general hospitals. Alien physicians were found to be concentrated in tuberculosis hospitals, where they accounted for almost one-half of the house staffs. They comprised over one-quarter of the house staffs of general nonteaching hospitals and almost one-quarter of the staffs of mental hospitals; however, they represented a much smaller proportion of the staffs of teaching hospitals—an average of 12%.

Regional Distribution.—Hospitals in 45 states and the District of Columbia reported intern and residency programs in operation during 1953-1954, and only 3 of these states—Mississippi, North Dakota, and Arkansas—were without the services of alien residents and interns. Owing chiefly to state differences in licensing laws and requirements, the alien physicians were concentrated in certain sections of the country. Over two-thirds of the aliens were in five states—New York, Ohio, New Jersey, Illinois, and Massachusetts. In New Jersey, 65% of the house staff positions were filled by aliens, and in New York, Illinois, and Ohio the comparable percentage was over 30 (table 3).

Year of Graduation.—The alien intern had been out of medical school longer than the citizen intern, on the average. More than 94% of the United States citizens who were first year interns in 1953-1954 completed medical school in 1953; in contrast, only 20% of the aliens were 1953 graduates. Similarly, for aliens serving in residencies, the median year of graduation was 1949, for citizens, 1950.

Age.—The typical alien house staff member was about two years older than his citizen counterpart. For the training year 1953-1954, the median age was 29 years for alien first year interns as compared to 26 for the citizen intern; however, among residents and fellows, the median age for both citizens and aliens was 28 years.

From the Health Resources Advisory Committee (Dr. Diehl, Dr. Crosby, and Mr. Kaetzel), Dean of Medical Sciences, University of Minnesota (Dr. Diehl); Director, American Hospital Association (Dr. Crosby); and from the Office of Defense Mobilization (Mr. Kaetzel).

TABLE 1.—*Aliens Serving as First Year Interns and Residents in Hospitals in the United States, 1950 to 1954*

Year	Interns		Residents		Total	
	No.	%	No.	%	No.	%
1950-51						
Total	6,237		18,205		24,442	
Alien	722	11.6	1,350	7.4	2,072	8.5
1951-52						
Total	6,783		16,135		22,918	
Alien	1,116	16.5	2,233	13.8	3,349	14.6
1952-53						
Total	6,990		16,231		23,221	
Alien	1,353	19.4	3,035	18.7	4,388	18.9
1953-54						
Total	7,705		18,191		25,896	
Alien	1,787	23.2	3,802	20.9	5,589	21.6

TABLE 2.—*Aliens on Medical House Staffs in Approved Hospital Training Programs, by Type of Hospital, July, 1953*

Type of Hospital	Total Staff	Aliens	
		No.	%
General	23,246	4,859	21
Teaching *	11,341	1,353	12
Nonteaching	11,905	3,506	29
Under 200 census	3,320	1,419	43
200-299	3,319	1,168	35
300-399	1,755	374	22
400-499	772	130	17
500-999	1,756	306	17
1,000	983	109	11
Mental	1,167	268	23
Tuberculosis	340	145	43
Special	1,143	317	28
Total	25,896	5,589	22

* Hospitals that serve as major teaching hospitals for medical schools.

TABLE 3.—*Aliens on Medical House Staffs in Approved Hospital Training Programs, by States and Territories, July, 1953*

State	Total	Aliens	
		No.	%
New Jersey	594	381	64
South Dakota	17	10	59
Montana	4	2	50
West Virginia	104	51	49
Hawaii	57	21	38
Ohio	1,675	541	32
Delaware	72	23	32
Rhode Island	102	32	31
Illinois	1,670	519	31
New York	5,001	1,480	30
Maryland	708	194	27
Massachusetts	1,432	363	25
District of Columbia	518	131	25
Arizona	62	15	24
Connecticut	475	108	23
Vermont	58	13	22
Missouri	868	173	20
Iowa	222	43	19
Minnesota	948	183	19
Virginia	437	80	18
Kentucky	230	41	18
Michigan	1,342	237	18
Utah	127	21	17
Canal Zone	26	4	15
Colorado	332	46	14
Texas	755	105	14
Wisconsin	362	50	14
Florida	237	32	14
Kansas	244	30	12
Washington	290	34	12
Pennsylvania	1,895	215	11
California	1,993	198	10
Tennessee	474	47	10
Georgia	350	30	9
North Carolina	370	32	9
Alabama	144	12	8
New Hampshire	62	5	8
Louisiana	542	37	7
New Mexico	15	1	7
Oklahoma	149	9	6
Oregon	201	11	5
Nebraska	130	7	5
Indiana	281	14	5
South Carolina	104	4	4
Puerto Rico	79	3	4
Maine	37	1	3
Arkansas	68	...	..
Mississippi	22	...	..
North Dakota	11	...	..
Idaho	...	...	..
Nevada	...	...	..
Wyoming	...	...	..
Total	25,896	5,589	22

Hospital Service.—Although alien residents were distributed throughout the specialty services, the distribution was uneven. The largest number, 747, was in surgery. This represented 18% of all surgical residents. Aliens constituted a smaller proportion of residents on internal medicine, 15%; radiology, 16%; and pediatrics, 16%. They represented a larger proportion of the residents on pulmonary disease, 41%; general practice, 38%; and pathology and anesthesiology, each 29% (table 4).

SUMMARY

Since the passage of the U. S. Information and Educational Exchange Act of 1948, there has been a great increase in the number of alien physicians taking postgraduate medical education in approved civilian hospital training programs. The total of 5,589 aliens in internships and residencies during the 1953-1954 training year represented 21% of the total in training. For the most part they were located in those general hospitals that do not serve as major teaching hospitals for medical schools. The highest proportion of aliens was found in the smaller nonteaching hospitals and tuberculosis hospitals. Over two-thirds of the aliens were in five states —New York, Ohio, New Jersey, Illinois, and Massachusetts. The average alien house staff member of 1953-1954 was about two years older than his citizen counterpart.

TABLE 4.—*Aliens Serving as Residents in Approved Hospital Training Programs, by Hospital Service, July, 1953*

Hospital Service	Total	Aliens	
		No.	%
Internal medicine	3,681	558	15
Radiology	907	144	16
Pediatrics	1,063	175	16
Surgery	4,072	747	18
Orthopedics	547	109	20
Psychiatry and neurology	1,846	379	21
Obstetrics and gynecology	1,537	323	21
Urology	361	79	22
Eye, ear, nose, and throat	666	163	24
Anesthesiology	650	191	29
Pathology	758	223	29
General practice	321	121	38
Pulmonary diseases	278	114	41
Other	1,504	476	32
Total	18,191	3,802	21

18 E. Division St. (Dr. Crosby).

Liver Biopsy.—Needle biopsies of the liver were obtained from 28 patients with extrahepatic biliary obstruction due to common duct stone, stricture of the common duct and neoplasm. The results of liver biopsies were compared with those of a battery of hepatic tests. The examination of liver biopsy specimens proved to be much more helpful in arriving at a diagnosis of obstructive jaundice than the combined results of cephalin cholesterol flocculation, thymol turbidity and serum alkaline phosphatase determinations. Moreover, a diagnostic biopsy of the liver could be obtained as early as within one week of the onset of jaundice. Needless to say, a procedure resulting in a definitive diagnosis at the earliest possible moment is of inestimable value. The early development of beginning periportal fibrosis was demonstrated by liver biopsy in some cases within only a few weeks of the onset of jaundice.—E. R. Movitt, M.D., and A. E. Davis, M.D., Extrahepatic Biliary Obstruction: Experience with Needle Biopsy of the Liver, *Annals of Internal Medicine*, May, 1954.

SUPPORT DURING LABOR

OUTLINE OF PRACTICE AND SUMMARY OF RESULTS FROM THE MOTHER'S VIEWPOINT

Herbert Thoms, M.D.
and
Ernestine Wiedenbach, M.A., R.N., New Haven, Conn.

In recorded history, assistance or support for the woman in childbirth has had a prominent place from earliest times. In all social orders, the midwife occupied a prominent position. In colonial America, she was often granted property and privileges that were given to no other member of the community. Today, the usefulness of the woman specifically trained for assistance in childbirth finds excellent expression in the midwife system of obstetric care so highly developed in certain European countries.

There is no question that the great change from obstetric care in the home to that in the hospital has brought untold benefits in safety for mother and child; this is an epoch in modern preventive medicine. However, it may be questioned whether certain aspects of the practice of obstetrics in hospitals may not be subject to speculation as to the quality of their usefulness. We refer to such practices as the separation of mother and infant (the central nursery system), the inability of the husband to stay with his wife during the labor period, the use of the general labor room in which a woman in labor is exposed to others in various stages of the process, and the use of heavy sedation to obliterate a greater part of the mother's memory of her childbirth experience.

Our own questioning of some of these things became manifest when, in association with the department of pediatrics, we established in 1946 rooming-in for mothers and infants as a regular part of the university teaching service. Shortly after this, in 1947, we had the good fortune to have as a volunteer member of our staff the late Mrs. Helen Heardman, a British physiotherapist extensively trained in methods of physiotherapy applied to obstetrics and the author of several books on the subject. Her demonstrations of what could be accomplished during labor and delivery in "unprepared" mothers was a revelation to all who witnessed her work. We pay tribute to her pioneer work in an important field of obstetrics.

This paper deals with one aspect of the preparation for childbirth program, namely, support of the mother during labor. Other aspects of the regimen have been discussed elsewhere.[1]

Our concept of support during labor we propose to give in outline, following which, we shall enumerate certain results from the mother's point of view. This latter information was obtained from questionnaires that mothers filled out just before their discharge from the hospital.

OUTLINE OF SUPPORT

Support during labor depends for its effectiveness not only on the sympathy and interest of those in attendance but on their comprehensive understanding of the physical and psychological aspects of the birth process and the details and importance of the preparation program. Effectiveness of support is also predicated on normal labor; that is, labor in a healthy woman in whom there is no cephalopelvic disproportion and no abnormal fetal position, in whom the soft parts are normally dilatable and the uterine contractions of proper regularity and of suitable force, and in whom delivery is possible without undue strain. A guiding principle is that expressed by Dr. Helene Deutsch,[2] namely, "to find a technic of delivery in which the psychic value of the mother's active participation in the process is taken into account, and to reunite mother and child as soon as possible after birth."

Practices that we follow include the following. 1. We try to make the mother feel important as an individual. We think of her as the central figure in the situation. We include her in our conversations with others in attendance. We consult her when it is appropriate to do so, and we introduce ourselves and others who may enter her room. 2. We try to give her confidence in her environment. We keep her informed of her progress and condition. We stay with her, and, if we have to leave even for a brief time, we tell her why we are leaving and when to expect our return. We make sure that she is able to call for attendance (call light or buzzer) should she wish it. We tell her in advance of examinations or special procedures that may be carried out. We try to be gentle, considerate, and protective of her when carrying out such procedures. 3. We institute measures to promote relaxation. These measures include privacy (individual room); quiet surroundings; a cool cloth to forehead, if desired; back rub, if desired; a clean, dry pad under her, as necessary; and warmth—hot water bag, if desired. 4. We instruct her in techniques that lead to effective performance. These techniques are abdominal breathing, relaxation, pelvic rocking, pushing with expulsion contractions, and panting at the proper time. 5. We endeavor to encourage her at all times by voice, look, and manner, always speaking with sincerity and remembering that the eyes can smile as well as the lips and that actions in themselves often speak with profound impressiveness.

A general outline of the support procedures that we endeavor to carry out on all mothers, whether prepared or not, can be summarized in the following statement. In our management of labor, we try to carry out a definite pattern, following several principles. First, we keep the mother informed of her progress. She is told about the position of her baby and of its progress through the birth canal. The devious explanation, the hushed tone, or the

From the Department of Obstetrics and Gynecology, the School of Medicine, and the Division of Obstetrical Nursing, the School of Nursing, Yale University.

1. Thoms, H., and Wyatt, R. H.: One Thousand Consecutive Deliveries Under a Training for Childbirth Program, Am. J. Obst. & Gynec. **61**: 205, 1951. Thoms, H., and Karlovsky, E. D.: Two Thousand Deliveries Under a Training for Childbirth Program, ibid. **68**: 279, 1954.

2. Deutsch, H.: The Psychology of Women: A Psychoanalytical Interpretation, New York, Grune & Stratton, Inc., 1945.

overly sympathetic attitude are all studiously avoided. Second, the mother is in a private room and not left alone unless she desires it, which may be the case very early in labor when she is napping between contractions. In any event, an attendant (nurse, medical student, or physician) is immediately available. If the mother desires, she may have her husband with her until she is moved to the delivery room. The physician sees her at proper intervals, especially during certain important periods in the labor. Third, in prepared mothers the techniques of relaxation and breathing are instituted when they are necessary. In unprepared mothers, an attempt is made to institute these techniques as far as possible. In the average labor, the time for this is usually when the external os is dilated about 4 cm. It is at about this time that the contractions are apt to be stronger and more regular. The relaxation position and abdominal breathing techniques can alleviate much of the woman's discomfort, which in turn will reinforce her confidence. If the husband is with his wife and has attended the classes for expectant parents, he knows something of what is going on. He is often helpful as a back rubber. This is done with the mother on her side in the general relaxation position and consists essentially of massage with firm pressure over the lower back. If at any time the mother's morale seems weakening, attention is immediately given. This attention may consist simply of encouragement, further instruction in relaxation, or the giving of a sedative. When the latter is given, the mother is told that it will help her relax, which it usually does.

The critical period of labor is at the end of the first stage when the cervix is almost fully dilated. This is the transition stage. It is at this time that the mother may show considerable anxiety and have more or less severe backache. Continuous attention and encouragement are important during this period. The mother is told that after 10 or 12 contractions she will experience relief. During this transition stage, sedation is often helpful or whiffs of gas may be given. Many women, however, pass through this period without too much discomfort, practicing relaxation during contractions and quietly dozing between times.

The relaxation and breathing techniques are also valuable in the second stage of labor. As each contraction finishes, the woman is instructed to take three or four breaths by abdominal breathing; this will assist her in continuing relaxation. She is told not to resist the expulsive effort of the uterus. She is kept informed as to the progress of the fetal head and is told that she will have a feeling of stretch during the actual delivery, also that soft panting breaths will prevent a too rapid delivery. Nitrous oxide or trichlorethylene (Trilene) is administered whenever the woman desires it, and infiltration anesthesia is used when episiotomy is done.

Most women who have been prepared and who have spontaneous births are quite aware of all that goes on during delivery, and immediately after this event they have a feeling of well-being and are inclined to be talkative. They are often quite interested in the expulsion of the placenta. Our practice is for the mother to remain in the delivery suite for at least one hour after the birth of her baby. The infant is usually put to the breast at some time during this period.

Experience has shown that in normal labor the period of greatest discomfort is almost universally at the end of the first stage, the transition period. Soon after this, as the head begins to descend, the picture usually changes markedly, and the woman who may have been fussing about her discomfort settles down to the task before her. Anesthetic agents not only are not withheld during the second stage but are sometimes urged on the woman who is having too much discomfort but not asking for help. Those who are in attendance at this time should be wary of misinterpreting the facies of effort as that of pain.

Certain points need further emphasis. We are convinced that a woman's sense of security is enhanced by not being alone when she is in active labor, that she prefers to be in a private room, and that for most women the presence of the husband adds greatly to the sense of assurance and comfort.

During early labor the mother is permitted to move about, to sit or lie in bed as she desires. At any time during the first stage of labor, if she is having more discomfort than she is willing to bear, the physician sits with her for a while to find the cause of her difficulty. Something is done about it—encouragement, instruction, sedation, etc. Attendants must not expect that all women who have attended classes are equally well prepared or that they will go through an ideal birth experience. It must be impressed upon many women that the need for sedation to combat discomfort or to aid in relaxation does not in any sense represent failure on their part. The woman who is anxious to do well often sets too high a standard of accomplishment for herself and as a result may become discouraged when things do not proceed as rapidly or as satisfactorily as she fancied they would.

QUESTIONNAIRE

A questionnaire was used in order to obtain some evaluation of the mother's own assessment of her labor and delivery. It is usually filled out a day or two before the mother goes home and is of course voluntary on her part. The content is seen in the complete report of a 23-year-old primipara, formerly a stenographer. Her child was born in December, 1953.

1. Describe how you felt, what happened, what the contractions (pains) felt like, what you wanted to do or have done for you.

(*a*) In the labor room.

"Between contractions I felt good. The contractions started with a slight pain, getting stronger, and then leaving. The nurse helped me do abdominal breathing when I thought I couldn't do it. I was not in the labor room too long until the contractions gave me a rather bearing down feeling."

(*b*) In the delivery room before your baby's birth.

"I had a definite bearing down feeling with my contractions. I liked the idea of a mirror. I watched almost everything. I feel I had one of the best experiences I'll ever have in life."

2. How did your labor start?

"The pains started as a small cramp, something like menstrual cramps."

3. What helped you during your labor and delivery?

"Abdominal breathing. Medicaments."

4. What did you mind during your labor and delivery?

No answer.

5. What were your first thoughts after the birth of your baby?

"I wanted to hold the baby. I was just relieved that it was all over."

(*a*) How did you feel?
"I felt just fine."

6. How did your delivery compare with what you expected?
"It was much easier than I expected and not so long as I expected."

7. If you have another baby, would you like to have it the same way as this one?
"Definitely."

8. Did you attend mothers' classes?
"Yes."

(*a*) What did you gain from the classes that helped you in your labor and delivery?
"I think the exercises helped me very much. Also the lectures we had in mother's classes gave me a clear picture of what would happen. That helped my mental attitude greatly. The tour of the hospital helped too. I knew exactly where I would go and what the equipment looked like. I believe the parent's classes helped, too."

9. We would value any suggestions you may have or any frank statement you would care to make that would help us in our guidance of mothers through childbirth.
No statement.

10. What is your impression of rooming-in?
"I think rooming-in is something all hospitals should have. There are many mothers with first babies who know nothing about caring for them. Rooming-in gives them a chance to learn how to take the best care of their babies. Also, a mother gets to know her baby before she goes home. Having the baby in the room with you helps pass the days quickly. My stay whizzed by. I would like to mention also that I received very good care during my stay."

RESULTS

Questionnaires similar to this one were received from 142 mothers; 83 of them had attended classes, and 59 had not. Of the mothers who attended classes, 29 were delivered by private physicians. Of those who did not, one was so delivered.

The group that did not attend is of particular interest for it is obvious from their answers that many of them have somewhat lower intellectual attainments than the majority of the women in the group that did attend classes. Their nonattendance at classes apparently was due to lack of interest. We recognize this as a challenge and suggest that a simpler and shorter form of class work may be desirable for such mothers. This would be aside from the regular program. The survey shows that of those who did attend (83), 76, or 92.7% of the mothers had a high school education. In the group that did not attend (59), 22, or 37.1%, had a high school education. In the group that attended classes, 79.5% of the mothers consulted a physician before the fifth month of pregnancy. Of those that did not attend, 30.5% said they consulted a physician before the fifth month; 15 did not answer.

In the group attending, the presence of the husband or relative was commented on in 77 instances in the 83 labors. It was emphasized as very helpful in 36 instances. In the group not attending, this was commented on in 47 instances in 59 labors and emphasized in 12 instances. One woman wrote, "So glad you love our husbands as much as we do"; this has its own message.

In answer to question 3, "What helped you during your labor and delivery?" 43 mothers who attended classes emphasized the confidence that was felt in the attention received, the presence of the husband, and the interest and helpfulness of the attendants. Thirty-seven mothers emphasized the help they received from practicing the relaxation and breathing techniques. Twenty-two mothers emphasized the aid received from the administration of medicaments. In the group not attending, 33 mothers emphasized the presence of the husband and help from attendants, 5 emphasized relaxation and breathing techniques, and 26 commented on medication. One woman stated tersely, "1. Husband, 2. Demerol, 3. Nitrous oxide."

In response to question 4, "What did you mind during labor?" the answers that seem significant for us are as follows. Of the women who attended classes, 7 minded episiotomy and stitches, 4 rectal examinations, 21 severe pain; 2 hypodermic injection, 2 noise, talking, etc. in delivery room, and one shaving with a dull razor. Of the women who did not attend, 4 minded episiotomy and stitches, 4 rectal examinations, 18 severe pain, and one noise, talking, etc. in delivery room. These expressions we believe, usually reflect some inattention or lack of skill on the part of attendants.

With regard to question 8, "What did you gain from classes that helped you in your labor delivery?" 58 of the mothers who attended emphasized the knowledge of the physiology of childbirth. Fourteen mothers emphasized that the tour of the obstetric division gave familiarity of the labor and delivery rooms. Forty-six women emphasized the relaxation and breathing techniques. The importance of classes as a shared experience was also recorded. Certain short answers were meaningful. One woman wrote simply, "Peace of Mind," another, "What to expect and how to relax."

Twenty-eight women in both groups were further questioned as to their reaction to the rooming-in arrangement. There was no unfavorable comment, and all were enthusiastic in its favor. One woman finished a rather lengthy discussion of the subject with, "It is just nice to have the baby near you to look at, too."

COMMENT

We believe the results of the questionnaire have been helpful to us in evaluating our efforts in carrying out the program. We are convinced of the usefulness of planned and integrated support for all women during labor and urge its consideration as part of all labor and delivery room practice. Those attending parturient women can be more effectual in their ministrations if they are willing to adopt a definite labor support regimen, thoroughly understood as a cooperative enterprise. Greater benefits can come from such an endeavor if the experience of physicians and nurses is shared in regularly held conferences. The mother's point of view is often greatly helpful in pointing out weak spots in the program.

SUMMARY AND CONCLUSIONS

We believe that support in labor should provide an atmosphere of friendliness, interest, understanding, and encouragement coupled with effective comfort-producing measures that will enable the mother to put forth her best effort in the birth of her child. Everyone who attends her contributes to the effectiveness of such support. This includes her husband or other family member, physician, nurse, medical student, student nurse, porter, aid, etc. The more each participates wholeheartedly and cooperatively in giving support, the better will be the mother's experience.

789 Howard Ave. (4) (Dr. Thoms).

IMPORTANCE OF GASTRIC MUCOSA IN MECKEL'S DIVERTICULUM

Edward J. Berman, M.D., Albert Schneider, M.D.
and
Willis J. Potts, M.D., Chicago

The object of this brief communication is to reemphasize some of the complications of Meckel's diverticulum, the majority of which are the result of ectopic gastric mucosa. The course of the embryologic mishap responsible for the persistence of this congenital remnant has often been reviewed; during early fetal life bits of gastric mucosa are occasionally misplaced in the diverticulum, and in this functioning gastric mucosa peptic ulcers develop. In a group of 25 consecutive cases of symptom-producing remnants of the omphalomesenteric duct, 4 patients had persistent umbilical sinuses that required excision and were found to extend to the ileum. Two patients had rupture of an ulcer in Meckel's diverticulum with all the signs of acute abdominal disease; two had strangulation obstruction due to a persistent strand of tissue extending from the diverticulum to the abdominal wall; and one had a Meckel's diverticulum associated with intussusception. In the remaining 16 patients, rectal bleeding was the cardinal symptom.

ECTOPIC GASTRIC MUCOSA

It is well known that gastric mucosa may be found in a Meckel's diverticulum. We are inclined to believe, after microscopic study of the diverticula associated with bleeding or local rupture of a peptic ulcer, that bleeding or rupture occurs only when gastric mucosa is present. There is a striking correlation between rectal bleeding from and perforation of a Meckel's diverticulum and the presence in it of gastric mucosa. Microscopic sections of Meckel's diverticulum without reference to symptoms, according to Gross in "The Surgery of Infancy and Childhood," show gastric mucosa in 53% of cases. However, when the cardinal symptoms are bleeding or perforation, over 90% of the diverticula contain gastric mucosa. We suspect that, if numerous microscopic sections are studied, resected diverticula that have bled will show gastric mucosa in nearly 100% of cases. In some instances, a small island of gastric mucosa was found and in it a tiny bleeding ulcer.

CLINICAL FINDINGS

Vague abdominal symptoms, such as those ordinarily ascribed to chronic appendicitis, are rarely due to a Meckel's diverticulum. If during operation a Meckel's diverticulum is unexpectedly found, it should be removed if conditions are favorable; however, exploratory operation primarily for the removal of a Meckel's diverticulum on a patient who has never had rectal bleeding will rarely be justified. The outstanding, and usually the only, symptom of the presence of a Meckel's diverticulum is bleeding from the rectum. The mother states that her child (probably from 1 to 6 years old, rarely over 12) has suddenly passed a large amount of blood. An analysis of the amount of blood passed and its color is of prime importance. If the stool containing blood was passed in the toilet bowl the mother will have overemphasized the amount of blood passed; even a teaspoonful of blood in the bowl will make the water red enough to frighten the mother. The color of the blood is equally important. Usually, the first bowel movement in an otherwise normal child will be formed and will contain some brick-red blood. The second movement likewise will be brick-red. Only if the bleeding is very profuse and persistent will bright red blood be passed. Occasionally, a perfectly well child may go into unexplained shock and after several hours become pale, weak, and listless—show all the signs of hemorrhage—although no blood has been seen. One should be suspicious of a hemorrhage from a Meckel's diverticulum; stool examination may provide the answer.

Whenever a mother calls and says her child has passed blood, every stool should be saved in order that the amount of blood passed can be estimated. A rectal examination done soon after the report of bleeding will often be of value because the gloved finger may withdraw some material which has the almost diagnostic brick-red color. If the patient is an infant every diaper containing stool must be saved and examined. One may well be led astray by incorrect observations by the mother. In one instance a mother reported recurring blood in the stool of her 2-year-old child with no other symptoms. Stool examination revealed the harmless cause; the child preferred beets to all other vegetables.

It is very important when evaluating blood in an infant's or child's stool to learn its relation to the bowel movement. If the blood is on the outside of the stool, if it is bright red, and if the infant cries when the stool is passed, the diagnosis will likely be a fissure of the rectum, common in infants but not in older children. If the child passes a normal stool, then in a few minutes requests to go to the toilet again and passes a few cubic centimeters of bright red blood, sometimes clotted, the child probably has a polyp in the rectum. Such a diagnosis is easily confirmed by a rectal examination or by the mother's report that something looking like a cherry will appear at the rectum after bowel movement and then disappear spontaneously. Polyps high in the rectosigmoid may be difficult to differentiate from a bleeding Meckel's diverticulum. The symptomless or slightly anemic child may suddenly pass a fair amount of blood from the rectum, and the blood may be somewhat dark because it has been retained in the rectum for some hours until it has the "brick-red" appearance of blood so characteristic of a bleeding Meckel's diverticulum.

Bleeding from the intestine in children may be caused by many conditions, such as intussusception, diffuse polyposis, ulcerative colitis, endemic diarrhea, Henoch's purpura, diarrhea (in the newborn), and a number of

From the departments of surgery and pathology of the Children's Memorial Hospital.

other less common causes. In all such cases associated symptoms and specific physical findings make the differentiation from a bleeding Meckel's diverticulum relatively easy. When a child with confirmed bleeding from the rectum but otherwise without symptoms is admitted to the hospital, only a few studies are necessary besides routine physical and laboratory examination including a rectal examination after an enema has cleared the rectum. A rectosigmoidoscopy is done to rule out polyps in the visible portion of the distal bowel, and a barium enema study to rule out polyps or other pathological condition in the colon. There is little use in wasting time and the patient's money by trying to demonstrate a Meckel's diverticulum with roentgenographic studies. In summary, one may say that if a child has passed considerable confirmed blood by rectum, if that blood has been dark red, and if further studies show no other cause for bleeding, surgical exploration usually is indicated.

SURGICAL CONSIDERATIONS

It is axiomatic that a child who has bled enough to lower the hemoglobin level is given a transfusion before or during operation, or both. The Meckel's diverticulum is usually found about 18 in. (42.26 cm.) proximal to the ileocecal valve. An acceptable procedure is to start at the ileocecal valve and inspect the small intestine from there proximally. If a diverticulum is not found after going well up the jejunum, the procedure should be repeated by gently stripping the intestine through the fingers. The virtue of this procedure is supported by events in the case of a healthy 4-year-old boy who suddenly passed a considerable amount of brick-red blood on two occasions. He was operated on, and the surgical report stated rather definitely that no Meckel's diverticulum was present. Another episode of bleeding followed, and he was admitted to the Children's Memorial Hospital showing definite signs of past hemorrhage. He was again operated on, and a Meckel's diverticulum, about the size and shape of a walnut, was found seemingly in the mesentery. After it had been freed from surrounding structures it was found that the diverticulum, typically attached to the antimesenteric border of the ileum, had been drawn against the mesentery, where it had been covered with a layer of adhesions that looked like part of the peritoneum of the mesentery. The adhesions were undoubtedly the result of previous inflammation. In this position the diverticulum had been mistaken at the first operation for an enlarged lymph node. The diverticulum is usually easily removed by clamping it transversely at its base with a toothed ductus clamp; after it is removed the stump is closed and inverted with a row of fine silk Lembert stitches. Occasionally, because of the size of the base of the diverticulum, it is necessary to resect a segment of small intestine. In such cases a simple end-to-end anastomosis is done.

Clinical Manifestations in Twenty-Five Patients with Meckel's Diverticulum

Clinical Manifestations	No. of Cases	No. with Gastric Mucosa in Diverticulum
Bleeding alone	13	11*
Bleeding and perforation	2	2
Bleeding with intussusception	1	0
Perforation	2	2
Umbilical drainage	4	1†
Strangulation obstruction	2	0‡
Intussusception	1	1

* One specimen not sectioned.
† One specimen lost; epithelium necrotic in one.
‡ Epithelium necrotic in both.

707 Fullerton Ave. (14) (Dr. Potts).

CLINICAL EVALUATION OF TUBADIL

E. R. Malia, M.D., M. A. Lucas, M.D., K. O. Burns, M.D.
and
R. J. Whitacre, M.D., East Cleveland, Ohio

The efficacy of *d*-tubocurarine as an adjunct to anesthesia has long been established. The ability of curare to relax striated muscle led to its use in control of muscle spasm following traumatic surgery [1] and in the treatment of neurological disorders.[2] Recently, *d*-tubocurarine was combined in a slowly absorbed menstruum and marketed under the trade name of Tubadil. Each cubic centimeter of Tubadil contains *d*-tubocurarine chloride pentahydrate, 25 mg.; oxycholesterol derivatives, 312 mg.; white wax, 20 mg.; chlorobutanol anhydrase, 5 mg.; and deproteinized peanut oil, 562 mg. The aqueous solution of curare is intended to provide a brief relaxant effect for only the period of surgery with no carry-over into the postoperative period. Tubadil, on the other hand, is designed to provide relaxation for spasm and resulting pain that become apparent some hours after the effect of the anesthetic has worn off.

The encouraging report of Fuller [3] prompted our use of this drug in a variety of surgical cases. This report deals with 100 patients who underwent anorectal surgery and who received Tubadil postoperatively and a control group of 100 similar patients who did not receive Tubadil. All patients were operated on after spinal anesthesia was administered.

CLINICAL STUDY

The average dose of Tubadil was 1.5 cc. (37.5 mg. of *d*-tubocurarine chloride) intramuscularly at the time of surgery or immediately postoperatively. This was followed by an additional 1.5 cc. in 12 hours. Extreme caution must be exercised when employing larger doses or more frequent administration of Tubadil. To empha-

From the Department of Anesthesia, Huron Road Hospital.
The Tubadil used in this study was obtained from Endo Products, Inc., Richmond Hill, N. Y.

1. Fuller, J. D.: Use of Slowly Absorbed Suspension of *d*-Tubocurarine Chloride in Traumatic Injury, J. A. M. A. **143**: 789 (July 1) 1950.
2. Jonez, H. D.: Management of Multiple Sclerosis, Postgrad. Med. **11**: 415, 1952.
3. Fuller, J. D.: Advantages of the Preoperative Use of Slowly Absorbed *d*-Tubocurarine, Anesthesiol. **13**: 370, 1952.

size this vitally important precaution further, the following case is cited. Preoperatively, a patient with a herniated intervertebral disk was given Tubadil, 2 cc. every 12 hours. After the fourth dose, the patient complained of difficulty in breathing. The physician who visited the patient reduced the dosage of Tubadil to 1 cc. Shortly after the fifth dose (1 cc.), the patient became apneic and required heroic resuscitative measures to prevent death. The total dose of Tubadil was 9 cc. (225 mg. of *d*-tubocurarine) in 48 hours. This instance should not incriminate the drug as dangerous or harmful per se, but rather serve to illustrate the hazard attached to Tubadil as perhaps with any other drug when used indiscriminately or in excessive amounts.

TABLE 1.—*Distribution of Patients According to Sex and Age*

	Patients in Control Group	Patients Receiving Tubadil
Total no. of patients studied	100	100
No. of men patients	53	54
No. of women patients	47	46
Average age, yr.	41.5	40.45

No change was made in premedication, postoperative narcotic orders, or routine supportive care ordinarily given to these patients. The patients were unaware of the purpose of the drug, and the nurses administered narcotics for postoperative pain as indicated in the usual manner.

In both the control series and the group receiving Tubadil, the patients were selected consecutively. A comparison of age and sex between the two groups, as shown in table 1, indicates there was an even distribution.

At first there was some concern that the use of Tubadil might increase postoperative urinary retention, so it was decided to keep an accurate account of the number of patients requiring catheterization. Actually the number of patients requiring catheterization was 14 less than in the control group. While this may not represent an unusually large decrease, it is significant that urinary retention was not increased. This encouraging result might be explained by the relief of severe perianal pain obtunding reflex spasm of the urinary sphincters.

TABLE 2.—*Average Number of Doses* of Narcotic per Day*

Postoperative Day	In Patients in Control Group	In Patients Receiving Tubadil
1	2.1	1.9
2	2.2	1.5
3	1.6	0.7
4	1.0	0.3
5	0.6	0.1
Total	7.5	4.5

* Average dose was 10 mg. of morphine sulfate subcutaneously.

The patients were observed over a five day postoperative period during which time the doses of Tubadil and narcotics were carefully recorded. Each patient was visited daily during the five day period. We were impressed that most patients who received Tubadil appeared much more comfortable than those in the control group, although this is difficult to record statistically because of the considerable difference in pain threshold among patients and the variance of surgeons in their operative technique and postoperative care. The most impressive effect of the drug in lessening narcotic demand (table 2) appeared after the second postoperative day.

Analysis of the narcotic demand substantiates our clinical impressions. There was an average total reduction of three doses of morphine in a five day period, as shown in table 2. Percentagewise this represents a decrease in narcotic demand of 38%. Additional and continued effect of the drug is evidenced by the third day, when 53% (table 3) of those receiving Tubadil required no narcotics as compared to 29% in the control group, and by the fifth day, when 93% as compared to 61% in the control group required no narcotics.

COMMENT

In this group of 100 patients who received Tubadil, there were no reactions or complications, except for transient diplopia and muscular weakness. Serious complications may occur with overdosage of the drug as is illustrated by a near fatality in a patient who received 9 cc. of Tubadil within a 48 hour period before surgery. Since patients who receive Tubadil usually have some impairment of muscle control, they should be assisted when out of bed to avoid injuries due to falls.

TABLE 3.—*Number of Patients Not Requiring a Narcotic*

Postoperative Day	Patients in Control Group	Patients Receiving Tubadil
1	1	6
2	9	18
3	29	53
4	39	77
5	61	93

It should be clearly recognized that a drug that contains 25 mg. of *d*-tubocurarine chloride per cubic centimeter is potentially dangerous, even though it is absorbed slowly when injected intramuscularly. Although we have not observed evidence of uneven absorption of Tubadil when administered intramuscularly in the proper dosage, the result of intravenous administration can be hazardous. This is illustrated in a report [4] that we received of a death following accidental intravenous administration of the drug. The clinical impression was that death resulted from respiratory paralysis caused by the curare component of Tubadil.

Since the encouraging results following the use of Tubadil in anorectal surgery, it has been used in other surgical procedures, such as surgery of the eye, orthopedic surgery, and general abdominal surgery.

CONCLUSIONS

In a series of 100 patients who underwent anorectal surgery, *d*-tubocurarine chloride in a slowly absorbed menstruum, Tubadil, was given intramuscularly with the intention of decreasing muscle spasm. A series of 100 patients not receiving the drug served as controls. The results of the study indicate that postoperative pain, as well as narcotic demand, was decreased when Tubadil was used. There is also some evidence that postopera-

4. Zintsmaster, J. L.: Personal communication to the authors.

tive urinary retention was decreased. Other than transient diplopia and muscular weakness, which may be expected of a muscle relaxing drug, the only complications that we are aware of were caused by overdosage or intravenous injection of the drug. Because of decreased voluntary muscle control, patients should be assisted when out of bed. Since one death has been reported following the accidental intravenous administration of Tubadil, extreme caution must be exercised to assure that the drug is given only by the recommended intramuscular route. To avoid serious complications, the dosage of Tubadil should be limited to 1 or 2 cc. (25 or 50 mg.), and the dose should not be repeated more than once after a 12 hour interval, except in rare or unusual circumstances and then only under the direct supervision of the attending physician. It is our clinical impression that Tubadil offers certain advantages in the postoperative management of the hemorrhoidectomy patient and that the drug is worthy of further study in other types of surgery.

RECENT ADVANCES IN CLINICAL ASPECTS OF POLIOMYELITIS

Russell J. Blattner, M.D., Houston, Texas

Anyone who has had experience with infections caused by the virus of poliomyelitis is aware of the many complexities encountered in delineating the clinical manifestations accurately. Difficulty in diagnosis, particularly differential diagnosis, is increased by pressure on the physician from a fearful public. Improved understanding of the disease in its many aspects will contribute to the peace of mind of all persons concerned, including the practicing physician.

The virus of poliomyelitis belongs to the group of infectious agents designated as obligate intracellular parasites. These agents require the environment of a living cell for growth and multiplication. Although the distribution of the agent may be widespread in the body of the host, the poliomyelitis virus does have a peculiar affinity for cells of the central nervous system, particularly those of the anterior horn of the spinal cord and its bulbar extensions. Careful study of the pathological picture produced by this viral agent indicates that many portions of the neuraxis may be involved without corresponding clinical involvement. While such observations make accurate correlation of clinical with pathological findings more difficult, they contribute to a better understanding of the fact that the distribution of muscle paralysis is characteristically spotty and haphazard and usually asymmetrical. There is no infallible method available at present for making a definite diagnosis of poliomyelitis during the earlier phases of the infection. The physician must rely on clinical judgment for interpretation and evaluation of the findings in each individual case. As a clinician gains experience with poliomyelitis, he becomes more aware of the protean nature of this variable clinical syndrome. However, certain clinical features of the disease are sufficiently consistent to merit some emphasis.

CLINICAL TYPES OF INFECTION

For convenience, infection with the virus of poliomyelitis in the human subject has been classified into four general groups: (1) silent infection, (2) abortive poliomyelitis, (3) nonparalytic poliomyelitis, and (4) paralytic poliomyelitis.

Group 1.—Silent infection is asymptomatic and inapparent and offers no clinical problems.

Group 2.—Abortive poliomyelitis, probably a common type of infection with this agent, is of considerable importance, particularly during an epidemic. The clinical picture, which is at best ill defined, is characterized by a brief, febrile illness associated with the symptoms of malaise, anorexia, nausea and vomiting, headache, sore throat, constipation, and nonlocalized abdominal pain. Clinical manifestations of coryza, cough, pharyngeal exudate, and diarrhea are not considered common findings in poliomyelitis. The practical value of attempting to recognize cases of abortive poliomyelitis, particularly during epidemic outbreaks of the disease, stems from the generally held clinical impression that bed rest during the febrile course and for a week or so after defervescence may be a factor in avoiding progression of the viral invasion.

Group 3.—Nonparalytic poliomyelitis constitutes a most interesting and important aspect of this viral infection. It is characterized by the presence of nuchal and spinal rigidity and by soreness and stiffness of the posterior muscles of the neck, trunk, and limbs. In cases of this type, headache, vomiting, constipation, drowsiness, fever, leg ache, and sore throat are often reported. On the other hand, "running nose," diarrhea, and cough are not found. The differential diagnosis between nonparalytic or preparalytic poliomyelitis and other conditions that simulate this disease is of considerable practical importance. Since many clinical conditions may be confused with nonparalytic or preparalytic poliomyelitis, even the most experienced, conscientious, and thorough clinician has difficulty in establishing an accurate differential diagnosis. The following table summarizes the clinical experiences of the Southwestern Poliomyelitis Respiratory Center in Houston, Texas, from 1950 to February, 1954. Of patients admitted to this center with the clinical diagnosis of acute poliomyelitis, about 15% proved on closer study to be suffering from conditions other than poliomyelitis. Almost 26% of cases were classified as nonparalytic poliomyelitis, and the diag-

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Professor of Pediatrics, Baylor University College of Medicine and Southwestern Poliomyelitis Respiratory Center.

Read in the Symposium on Recent Advances in Poliomyelitis before the Joint Meeting of the Section on Experimental Medicine and Therapeutics and the Section on Internal Medicine at the 103rd Annual Meeting of the American Medical Association, San Francisco, June 24, 1954.

Tabulations included in this report were prepared for a teaching syllabus by the staff of the Southwestern Poliomyelitis Respiratory Center and are reproduced with the permission of Dr. William A. Spencer, Medical Director.

This paper was discussed by Dr. William A. Reilly, San Francisco, and Dr. James F. Bosma, Salt Lake City.

nosis in these cases was not changed to paralytic poliomyelitis on the basis of a one year follow-up. The remainder presented clinical manifestations of paralytic poliomyelitis. Conditions other than those involving the central nervous system occurred in 58 cases and included pneumonia, influenza, rheumatoid arthritis, rheumatic fever, scurvy, and hysteria. Of those patients with central nervous system involvement, 26 were considered to have encephalitis, type as yet undetermined; 6, mumps meningoencephalitis; 9, bacterial meningitis; 9, virus encephalomyelitis (infectious neuronitis); 3, lymphocytic choriomeningitis; 3, brain abscess; and 1, syphilis. Other conditions with central nervous system involvement that were confused with poliomyelitis included 11 other cases of infection, 4 cases of brain tumor, 3 of cerebral hemorrhage, and 1 of spinal cord tumor.

*Cases Initially Diagnosed as Acute Poliomyelitis, 1950 to February, 1954**

Final Diagnosis	No.	%
Not poliomyelitis	151	15.2
Nonparalytic poliomyelitis (1 year follow-up)	256	25.7
Paralytic poliomyelitis	587	59.1
Total	994	100

* Prepared for Teaching Syllabus by the staff of the Southwestern Poliomyelitis Respiratory Center and reproduced through the courtesy of Dr. William A. Spencer, Medical Director.

Proper evaluation of such an interesting group offers a real challenge to the clinician. While accurate diagnosis is impossible in certain of these cases, careful evaluation of the findings for each case tends to turn the thinking of the physician and of the parents away from the diagnosis of poliomyelitis. Avoidance of unnecessary admission to a hospital, particularly during epidemic periods, may spare the patient exposure to other diseases, including poliomyelitis. In patients with a history of increasing vomiting and intensification of soreness and stiffness of the neck, trunk, and limb muscles, the diagnosis of poliomyelitis must be seriously considered. Complete examination of the undressed child is necessary. Diagnostic maneuvers include evaluation of the patient's efforts to assume the sitting position, demonstration of nuchal and spinal rigidity, observation of the typical tripod sitting position, flexing of the chin on the chest, and testing of ability of the patient to kiss his knees. All these diagnostic maneuvers are used to demonstrate authentic cervical and back rigidity. The Brudzinski and Kernig signs and demonstration of head drop with the patient lying in the supine position may prove useful.

Demonstration of back rigidity in the young, uncooperative patient may be difficult. The fingers of the palpating hand are placed along the spines of the lower thoracic and lumbar vertebrae, and the back of the patient is extended gently with the patient lying in the prone position. In a normal child, even when he is uncooperative and struggling, progression of flexion of the spine is evidenced by differential elevation of the fingers along the spinal processes as the back is gradually extended. In contrast, on examination of the patient who has back rigidity, the examiner feels all the palpating fingers raised together in a single plane. The back feels "poker-like." This sign, while not always elicited satisfactorily, is often sufficiently suggestive to arouse suspicion that meningeal irritation is present and to warrant diagnostic lumbar puncture.

Other findings that suggest virus invasion include the presence of hyperesthesia and muscle tenderness and changes in the superficial reflexes, such as the cremasteric and upper and lower abdominal reflexes. The presence of signs and symptoms suggesting central nervous system involvement warrants the performance of a lumbar puncture. This procedure should be done under optimal conditions so as to avoid trauma and contamination. Characteristic findings include increased cell count (more than 10 cells per cubic millimeter, and usually less than 500 total), with early predominance of polymorphonuclears and later predominance of lymphocytes, and normal glucose level. Protein level is normal (35 to 40 mg. per 100 cc.) or may be slightly elevated early in the course of the infection. Within two to three weeks the protein level may rise to 300 mg. per 100 cc. Great variability in cell count has been reported. Pleocytosis may be lacking entirely, or the cell count may on occasion be elevated to 1,500 to 2,000 cells. Nevertheless, a fairly consistent pattern has been associated with poliomyelitis virus infection of the central nervous system, and it is of considerable diagnostic importance. A tabulation of the diagnostic criteria follows.

Suggestive
- Slight fever
- Meningism: stiff neck, stiff back
- Head drop
- Tripod sitting position
- Headache
- General malaise
- Vomiting or gastrointestinal symptoms
- Muscle pain or tenderness
- Reflexes normal or occasionally hyperactive

Confirmatory
- Flaccid muscle paralysis
- Absence of tendon reflexes in affected areas
- Spinal fluid showing pleocytosis, 10-500 cells; elevated protein level; normal glucose level; no bacteria; polymorphonuclears early, lymphocytes later

Positive
- Isolation of virus
- Rising titer of antibody level (virus neutralization test)

Group 4.—Those who become paralyzed constitute a clinical consideration of major importance. Scrutiny of the reflex patterns is of considerable prognostic significance. Changes in the deep tendon reflexes, either increase or decrease, forewarn of impending paresis of the muscles involved. Definite loss of reflexes, especially when all means of reflex reinforcement are used, is convincing evidence of paralytic involvement. The distribution of paralysis is asymmetric, involving several muscles of the same segment, and is characteristically spotty.

Once the diagnosis of paralytic poliomyelitis has been made, the question arises whether the involvement will progress and the patient become more acutely and, possibly, critically ill, and especially whether more serious manifestations of poliomyelitis, such as respiratory complications, will develop. Certain clinical findings are suggestive. Patients in whom the process goes on to muscle paralysis of varying grades of severity often have a flushed face accompanied by perioral pallor and a persistent but usually not high fever that tends to be sus-

tained at a level without marked diurnal variation. A septic fever is not considered characteristic of poliomyelitis infection. The conjunctivas are often suffused. The patient is apprehensive and seems frightened out of proportion to what might be expected from unfamilar procedures such as laboratory tests and separation from parents. These patients often have a "worried" expression, and they seem to have a feeling of foreboding. Cortical hyperirritability may be manifested by spontaneous, jerky movements that are often observed during sleep, which is usually fitful and restless. Some patients show definite fibrillary twitching of muscle bundles. This is regarded as an important forewarning sign of progressive muscle involvement. Opsoclonia, a form of nystagmoid movement of the eye, is an interesting sign that has been observed in some patients admitted to the Southwestern Poliomyelitis Respiratory Center. It is characterized by an oscillatory inability to fixate and is accentuated on extreme lateral gaze. The slow and rapid component of ordinary nystagmus is not seen. This ocular abnormality is not associated with permanent injury to the eye but is often associated with progression of paralysis and is regarded as a sign of prognostic import. While the Babinski sign may be present early, its persistence for more than a few days is usually accepted as evidence that one is not dealing with acute poliomyelitis infection.

Progression of the disease process, which is of paramount importance at this stage in the clinical syndrome, is variable. In general, further involvement occurs while the temperature is still elevated, and progression of paralysis does not continue for more than 48 hours after the temperature has reached normal. Persistent fever that cannot be explained by factors other than poliomyelitis, such as infection by secondary bacterial invaders, must be regarded as a serious prognostic sign. The progressive signs of paralytic poliomyelitis with serious muscle involvement may be summarized as follows:

Suggestive
- History of unusual exertion during prodromal symptoms in adults
- History of rapid onset of flaccid paralysis
- Presence of cranial nerve nuclear involvement (bulbar), especially lower cranial group
- Distribution of paralysis with involvement of the deltoid muscles
- Prostration
- Irritability and apprehension
- Higher temperature elevation
- Erythema or florid appearance
- Tachycardia
- Hypertension
- Nystagmoid ocular movements on extreme lateral gaze

Confirmatory
- Definite evidence of breathing difficulty
- Change in sensorium, severe apprehension, excitement, and confusion
- Swallowing difficulty
- Unconsciousness
- Cyanosis
- Peripheral circulatory collapse
- Extreme hyperthermia
- Pulmonary edema
- Myocardial failure

In evaluating the clinical course of these patients, the clinician is constantly on the alert for early signs of such serious complications. Any change in voice, especially toward nasal quality, must be regarded with suspicion. Progressive development of an ineffectual cough and evidence of difficulty in swallowing are early and important signs of more serious trouble to come. When early signs of breathing difficulty are recognized, the clinician and his staff are frequently hard pressed to keep abreast of the rapid changes in the clinical picture, and keen clinical judgment is needed to decide exactly when mechanical breathing aids are to be used for optimal therapeutic results. The following criteria for clinical determination of types of breathing difficulty have served admirably as a working guide for physicians at the Southwestern Poliomyelitis Respiratory Center.

Paralysis of the Breathing Muscles
- Shallow and rapid regular respirations
- Diminished movement of diaphragm and intercostal muscles (demonstrable by fluoroscopy)
- Decreased duration of phonation, as in counting
- Decreased vital capacity and resting tidal volume (demonstrable by basal metabolic rate machine)
- Dilation of the nares
- Preoccupation with breathing effort
- Use of accessory neck muscles
- Dyspnea

Obstruction of the Airway
- Bulbar involvement
- Mucus accumulation in the oropharynx
- Inability to swallow
- Shallow, often irregular respirations (fear of aspiration)
- Audible rhonchi (stethoscope bell over mouth)
- Inspiratory stridor with vocal cord paralysis

Involvement of the Respiratory Center
- Irregular, rapid, incoordinate, ineffectual respirations, with occasional deep breath and periods of apnea
- Usually associated with signs of central involvement:
  - Hyperthermia
  - Hypertension, severe
  - Peripheral circulatory collapse and vasomotor instability
  - Cardiac dysrhythmias

Fluoroscopic examination of the patient has been found helpful in evaluating respiratory efficiency, particularly in the case of infants and small children. The progressive signs of inadequate ventilation, resulting in hypoxia and carbon dioxide retention, follow.

Clinical
1. Restlessness, apprehension, anxiety, sleeplessness or lethargy, and change in sensorium
2. Florid appearance, increasing pulse rate, progressive or sudden elevation of blood pressure, constricted pupils, and profuse sweating
3. Dyspnea, intermittent cyanosis, occasional hallucinations, and panic reactions
4. Persistent cyanosis, pallor, circumoral twitching, and coma

Laboratory
1. Rising alveolar $CO_2 > 5.5\%$
2. (Compensation) Rising venous CO_2 combining power > 60 vol. %
3. Arterial anoxemia $< 94\%$ saturated hemoglobin
4. (Decompensation) Falling blood pH < 7.4

COMMENT

The physician's enormous responsibility in making a diagnosis of poliomyelitis, especially during epidemic outbreaks, is apparent to everyone who has had experience with this difficult disease. The best therapeutic course is often hard to follow, and the physician is constantly harassed by the problems of making crucial decisions. Since fatigue, excitement, and emotional strain are not good for the patient, it seems reasonable to urge that patients with nonparalytic or uncomplicated paralytic poliomyelitis be kept at home for treatment if at all possible. Such a policy would also conserve hospital and

financial resources. During the period when the infection is progressing it may be extremely difficult, if not impossible, for the physician to judge whether his patient requires special help in the hospital. Conscientious vigilance on the part of the parents and the physician should serve to inform the physician of the appearance of danger signals in sufficient time for safe transportation of the patient to a medical center if special treatment is needed. Each case requires individual consideration, and the physician in charge must assume responsibility for directing the therapeutic plan in every instance.

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RECENT ADVANCES IN THE TREATMENT OF POLIOMYELITIS

John E. Affeldt, M.D., Hondo, Calif.

While our horizons are bright with the prospects of preventing and modifying paralytic poliomyelitis, we are faced with the grim fact that in this country there are thousands of persons now undergoing treatment for their residual disability and 1,500 still residing in respirators of one kind or another, and that based on past experience there will probably be at least 30,000 new cases this season. It is also important to note that as yet there is no known drug or treatment technique that will substantially alter the course of the disease, once it has been clinically established. However, the combined efforts of physicians, engineers, manufacturers, physical therapists, occupational therapists, nurses, physiologists, technicians, research groups, and volunteer organizations dedicated to support such work have produced many advances in therapy that accomplish better end results. These advances can be roughly divided into two groups, those that reduce the mortality rate and those that attempt to restore function as completely as possible.

REDUCTION OF MORTALITY RATE

The advances responsible for the reduction in mortality include the early recognition and treatment of impaired vital functions, the treatment of pulmonary complications, and the establishment in smaller communities of poliomyelitis teams. Life is threatened only in those patients with involvement of vital functions such as respiration and circulation. A mortality rate based on all poliomyelitis cases, including the nonparalytic and mildly paralytic, is not very meaningful because it is likely to reflect a high proportion of the nonparalytic cases reported, but if based only on those with involvement of vital functions, then the figures will bear a relationship to the severity of the disease and the efficacy of treatment. Over the past few years, these advances have reduced the mortality rate in serious cases from over 50% to less than 20%, and in some places as low as 13%.

Only a few years ago the tank respirator was used as a last resort, and the poor results were used to prove that it was dangerous; today it is used early, before respiratory failure develops. One no longer waits for dyspnea, tachycardia, and cyanosis. Routine measurement of the vital capacity on admission gives a quick check on the status of the respiratory muscle involvement.[1] Serial measurements will reveal progressive involvement and allow putting the patient in a respirator when the vital capacity drops to approximately 30% of predicted normal. Better understanding of the mechanics of respiration and the physiological effects of the respirator have led to more rational and successful use. The desired tidal volume and minute volume can be predicted by one of several methods: by use of data obtained on normal subjects,[2] by use of a formula requiring measurement of oxygen consumption,[3] or by use of a nomogram prepared from metabolic tables.[4] Spirometers, basal metabolism machines, gas meters, or special ventilation meters [5] are available to measure tidal volumes, thus allowing the required setting of the respirator pressure and rate. Greater accuracy in measurement and control is available by determining the alveolar carbon dioxide concentration. This is now feasible with the infrared carbon dioxide analyzer, which gives continuous direct analysis sufficiently rapid to follow each breath accurately.[6] The use of these methods helps prevent hypoventilation and hyperventilation; the undesirable physiological effects of hypoventilation are well known, but the effects of hyperventilation are not as well defined or understood.

The nursing care of the patient in the tank respirator has been greatly facilitated by means of a positive-pressure attachment to the mouth or tracheostomy,[5] or by means of a dome covering the entire head. This allows opening of the respirator for necessary procedures while maintaining ventilation for the patient. A few years ago, tracheotomy was a subject for debate; today it is widely accepted and advocated. The disagreements now center around when and how many times to perform the operation, rather than whether it should be done. Establishment of a tracheostomy early, before an emergency develops, has reduced the incidence of complications. Experience has proved the need for careful suctioning and care of the tracheostomy, including humidification of

From the Department of Internal Medicine, College of Medical Evangelists, Los Angeles, and from the Rancho Los Amigos Respiratory Center for Poliomyelitis, Hondo, Calif.

Read in the Symposium on Recent Advances in Poliomyelitis before the Joint Meeting of the Section on Experimental Medicine and Therapeutics and the Section on Internal Medicine at the 103rd Annual Meeting of the American Medical Association, San Francisco, June 24, 1954.

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inspired gas, in order to prevent or minimize plugging and crusting. A variety of attachments to the tube are available for delivering humidified air, oxygen, aerosols for medication, and positive pressure for ventilation.[5] The ability to secure an adequate airway and to provide adequate ventilation has allowed differentiation of the causes of circulatory failure and shock into those due to viral action in the medulla or myocardium, those secondary to asphyxia, and those secondary to the effects of the respirator. The differentiation of those due to asphyxia from those due to the respirator is most important, for the treatment is quite different. If shock is present, increasing the intratank negative pressure will increase the shock, whereas the addition of intratank positive pressure on expiration may reduce the shock by increasing venous return to the heart.[7] The use of arterenol by continuous intravenous drip may sustain the peripheral blood pressure long enough to allow the return of a damaged circulatory center.

After the immediate results of viral action, the next complications most likely to threaten life are pneumonitis and atelectasis. The ability to culture and to test the antibiotic sensitivity of the causative organisms allows the rational selection and use of an increasing array of effective antibiotics. However, their frequent and prolonged use soon results in the predominance of organisms such as Pseudomonas aeruginosa and Proteus vulgaris, which are relatively insensitive to most available drugs. Pulmonary atelectasis is a frequent and major complication requiring aggressive therapy. This complication is being attacked quite successfully by a number of techniques, each of which and various combinations of which have their advocates; the spectrum ranges from nebulization to tracheotomy. A technique for nebulization producing supersaturated air, and the incorporation of various drugs such as bronchodilators, antibiotics, detergents, and trypsin[8] are proving to be useful, both prophylactically and therapeutically. Included with this should be systematic rotation of the patient, intermittent Trendelenburg position, and periodic deep breaths for lung expansion.

If these measures are not successful, bronchoscopy with or without direct instillation of trypsin may succeed; a recommended dose of trypsin is 250 mg. of Tryptar in 5 cc. of Sorenson's buffer (anhydrous dibasic sodium phosphate 6.6 mg. per cubic centimeter, anhydrous monobasic potassium phosphate 2.7 mg. per cubic centimeter in water for injection, pH 7.1). If this fails and a tracheostomy is not present, one should be provided. Besides these procedures, it is most important to provide a method for inducing a good cough. Most of the techniques currently in use are unsatisfactory. The thorax can be manually compressed during expiration. A deep breath can be achieved by increasing the intratank negative pressure. This is accomplished by attaching a vacuum cleaner to the porthole. When the negative pressure is 30 to 40 cm. of water, the bed pan port should be opened with a jerk to allow rapid passive expiration. More recently, a device has been developed which produces up to 40 mm. Hg positive pressure to the airway and by means of a selenoid valve then suddenly switches to 40 mm. Hg negative pressure.[9] This technique provides the best mechanical cough available thus far and promises to be a great help in the treatment of atelectasis, as well as an effective means of prophylaxis. Further improvement and developments along these lines can be anticipated.

It is obvious that the new and rapidly changing techniques and equipment cannot be readily available or even familiar to all practicing physicians. This is especially true in communities where only an occasional case of severe poliomyelitis occurs; it is in such areas that mortality runs highest. A number of communities have recognized this situation. They have been able to meet it and reduce their mortality to levels comparable with mortality levels in medical centers by organizing poliomyelitis teams among their local physicians. A team usually consists of an anesthesiologist to supervise the respiratory problem, a man to supervise the medical problems—such as pulmonary, circulatory, renal, and nutritional—and a man to supervise the orthopedic care. A general surgeon or otolaryngologist should supervise the tracheotomy. The functions of this team are to give special interest to the problems and techniques in poliomyelitis, to develop and implement consistent policies of treatment, and to accept the responsibility of caring for these patients. The cumulative experience of men on the team provides the know-how to solve acute problems and to achieve a greatly improved mortality rate and final end result.

RESTORATION OF FUNCTION

After the patient has survived the acute phase and become medically stabilized, the emphasis of treatment is shifted from life saving to function restoration. This shift in emphasis is more involved and complex than a superficial glance indicates. In the acute phase, all activity must be rigidly disciplined and centered on life saving, forcing curtailment of many desirable and long-range activities that are at that time secondary. In the restoration phase, a different philosophy prevails in which the discipline is directed toward the attitudes and actions of the patient and staff to provide strong motivation and the will to work for recovery. During this phase, the patients and their families must begin the long and difficult task of picking up the pieces to make the best of what is left. It is essential to have strong direction and coordination of the team in order to formulate a realistic goal and to progress in an orderly fashion toward it and also to prevent overactivity with resulting fatigue, wasted activity in nonproductive areas, false hopes by the patient, and conflicting statements on prognosis. Experience indicates that it is difficult for one staff to carry the two philosophies simultaneously, for it is inevitable that the life-saving philosophy will dominate; therefore separate staffs for these functions will give best results. While these staffs are basically separate in terms of responsibility, the greater the degree of cooperation and mutual understanding the better will be the final result.

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9. Barach, A. L.; Beck, G. J., and Smith, W.: Mechanical Production of Expiratory Flow Rates Surpassing the Capacity of Human Coughing, Am. J. M. Sc. **226**: 241-248 (Sept.) 1953.

The Lungs.—Once the medical emergency is over, it is time to begin efforts to decannulate the tracheostomy and to remove the patient from respiratory equipment. In some patients, it may be possible to accomplish this in a very short time, but in others it may require many months, and in some cases it may be impossible. The tendency is to procrastinate and leave patients in respiratory equipment too long; early and continued efforts should be made to get them out. Serial vital capacity determinations will indicate the progress of the respiratory muscles and can be used as a guide in removing equipment. A decrease of the vital capacity may indicate that the patient is being pushed too fast. Improved design and better understanding of the cuirass respirators (formerly called chest or portable respirators) have greatly increased their usefulness. At present there are two basic designs commercially available. One is a shell covering the chest and a bit of the upper abdomen, known as the chest cuirass; it is approximately 47% as efficient as the tank respirator as regards ventilation. The other is a shell covering the chest and abdomen, known as the chest-abdomen cuirass; it is 61% as efficient as the tank respirator.[10] As in using the tank respirator, it is essential to estimate the desired ventilation and then be sure, by actual measurement, that it is being achieved. This is even more important with the cuirass respirators than with the tank, because of their lesser efficiency. This is the reason cuirass respirators are not advocated in the acute phase. The efficiency of the respirator is an extremely important consideration, as not every device will adequately ventilate every patient. However, the fit and comfort of the cuirass is equally important, for regardless of its ability to ventilate, if it is uncomfortable, the patient will not and cannot wear it. It is best to have available both types so that individual fitting can be made to select the respirator most advantageous to a specific patient in a particular situation.

Rocking beds have also been improved by increasing their range of inclination; simplification of their mechanical features has decreased the problem of maintenance. Their ventilatory efficiency is close to that of the cuirass respirators; so the same principles of usage apply. An occasional patient will have motion sickness, but fortunately such difficulty is rare. The ventilatory efficiency of any respirator is not related to the degree of impairment of the respiratory muscles; it is related to the compliance and resistence of the respiratory system [11]; therefore the cuirass respirator or rocking bed may give adequate ventilation to a patient with complete muscle paralysis or a vital capacity of zero. There are many important advantages of these respirators over the tank. They allow freedom of motion and positioning of the patient, which is desirable for preventing deformities. They allow access to the patient by nurses, physical therapists, occupational therapists, and physicians. The cuirass respirator further allows a patient to practice walking, to sit in a wheel chair, or to travel in an automobile; all of these activities are extremely helpful psychologically. Pressure breathing devices are also available that attach to the cuirass pump and deliver pressure to the tracheostomy or to a pipe-stem type of mouth piece. This allows even greater mobility of the patient for short periods of time.

Glossopharyngeal breathing [12] has rapidly developed from its initial period as a curiosity into a very important substitute breathing technique that requires no mechanical equipment. The patient forces air into his lungs, using his tongue as a pump; a series of tongue strokes, followed by passive expiration, provides adequate ventilation and enables patients with essentially no breathing ability to get out of a respirator for hours at a time. This method of breathing allows patients who could barely breathe adequately for short periods when lying flat to get up into a wheel chair or automobile, or even to begin standing and walking. It can increase their vital capacity from essentially zero to 50 or 60% of predicted normal or, in some cases, to the full predicted value. This ability to take a deep breath periodically provides physiological lung and thoracic stretching and a fair cough. Patients cannot use this method of breathing while asleep as it is not involuntary or automatic, but it becomes semiautomatic while patients are awake in that they do not have to concentrate on it or even think about it. The length of time they can breathe by it varies considerably, and they can increase their endurance with practice. The ability to breathe spontaneously for even a few minutes offers an important safety factor to the patient dependent upon mechanical equipment for every breath; the development of this technique is a great boost psychologically.

The Extremities and Trunk.—The developments described thus far have been designed to get patients as free of respiratory equipment as possible. Another area of important development has been in restoring function to the extremities and trunk by treatment techniques, newer surgical procedures, functional bracing, and prostheses. For the past several years, a company in California has been experimenting with the development of prosthetic devices to provide motion for paralyzed upper extremities. One of the most important contributions to this field is the handy-hook, patterned after that used in most upper extremity amputation prostheses and controlled by a shoulder harness cable housing. The device is light and simple, it is especially effective since the patient with poliomyelitis retains normal sensation and joint movement. The company has also developed several other devices to aid in elbow and shoulder flexion. For some patients who have no functional muscle power of the shoulder, devices are attached to wheel chairs or to a functional lower extremity to activate the upper extremity; this development appears to offer tremendous possibilities in the rehabilitation of patients with severely paralyzed upper extremities.

For many years the transfer of tendons to new insertions has been done with rather remarkable success. This has recently been extended to the restoration of intrinsic musculature of the paralyzed hand, particularly of the first dorsal interosseus muscle. A new field in transplanting motor power has been presented and is based

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on the concept that a muscle may be freed at both ends and shifted to a new location where it will be successful in reinforcing weak function.[13] The most successful procedure so far has been in transferring a pectoralis major muscle into the arm to act as an elbow flexor. In this procedure, transplantations can be done with considerable shifting of position, providing the nerve supply and blood supply are left intact.

There is increasing emphasis on functional splinting of the extremities, using rubber bands to assist motion. For example, in the hand that has flexors but no extensors, an outrigger is made on a hand splint with attachments to the fingers to permit active flexion and passive extension. In this way activity of the hand is increased and recovery is hastened. The trend is to do earlier bracing in both the upper and lower extremities. This has several advantages: deformities are prevented, the motion that is present can then be used in a functional position, and weakened muscles are protected from overuse so that their recovery is hastened.

DEVELOPMENT OF REGIONAL CENTERS AND HOME CARE

The increased incidence of poliomyelitis, the greatly reduced mortality, and the many advances in treatment techniques and equipment available all add up to the obvious need for experienced teams to care for poliomyelitic patients. A few years ago the Medical Department of the National Foundation for Infantile Paralysis made a survey of the distribution of severely involved patients in respirators throughout the country. They found that the patients in respirators were widely scattered, usually only one or two patients in one hospital. They were receiving special nursing around the clock for months and years, and little, if any, attempt was being made to see if they could be removed from tank respirators. The situation was poor medically, socially, and economically.

The national foundation's solution to this problem was the development of nine regional respirator centers, strategically located throughout the United States and connected with teaching hospitals and research centers. They were assigned the responsibility of providing the best of care, research into the pathological physiology, development of improved equipment and treatment techniques, and a source of education for related professional fields. These centers have proved advantageous in many ways. The grouping of patients has allowed teams of men to gain needed experience to improve present methods of treatment and develop new techniques. The socialization of patients has increased their motivation and has given them confidence to try things formerly believed impossible. It has greatly reduced the cost of care, saving an estimated $3 for each dollar spent. The developments being made at the centers will carry over into other diseases and fields of medicine.

Despite all the progress listed, there still remains a certain residual group of patients who will require mechanical respirators of one form or another for years to come. Essentially, there is no need to commit patients to hospitals for the rest of their lives because of this. It has become apparent that they can be successfully cared for at home on a mutually advantageous basis. This is not easy to accomplish, but if all concerned are willing to make the effort, it can be done. It requires careful staffing, considering all aspects of the patient's needs and environment. Mechanical, social, nursing, and medical support must be provided. The cost of such care is one-third to one-half that of hospitalization, and provides a far more favorable social environment and makes available needed hospital space for newer patients. The parent, although in a respirator, can provide stability and guidance to the family. The child can be returned to the loving care of the parents. The severity of the case is no limitation; only the medically unstable patient cannot be safely cared for at home. At the Rancho Los Amigos Respiratory Center, we have developed a home care department; there are at present close to 70 respirator patients receiving service from this department within the County of Los Angeles.

SUMMARY

Several important advances have been made recently in the treatment of patients with paralytic poliomyelitis. The mortality rate has been reduced by increased use and understanding of tracheotomy and mechanical respirators of various types; by improved treatment of medical complications such as pneumonitis and atelectasis with antibiotics, trypsin, nebulization, and mechanical coughing devices; and by the development of local professional teams in smaller communities. There has been great improvement in techniques and devices for restoration of function, such as glossopharyngeal breathing, muscle transplants, functional bracing, and prostheses. Nine regional respirator centers have been established throughout the United States by the National Foundation for Infantile Paralysis, with the specific assignment of providing evaluation, care, and rehabilitation of patients and providing related research and education. Programs have been developed to send patients who must use respirators to their homes with adequate support to insure all of the advantages of the hospital, plus those inherent in the home situation.

Rancho Los Amigos.

13. Schottstaedt, E. R.; Larsen, L. J., Jr., and Bost, F. C.: A Report on the Orthopaedic Surgeons' Meeting: Mystery of Transplanting Muscles Attacked, Hosp. Topics 32: 16 (March) 1954.

Multiple Births.—In the births registered in the United States during the period 1933-1950, twins occurred in about . . . one out of every 92 confinements. Triplets occurred once in about 9,600 confinements, and quadruplets once in about every 657,000 confinements. The age of the mother has a considerable bearing on the chances of . . . plural confinement. The relative frequency of multiple births is least among mothers under age 20, increases with advance in age to a maximum for mothers in their late 30's, and then falls off toward the end of reproductive life. For teen-age mothers, the chances that a confinement will result in twins or triplets are only six in 1,000. For those aged 35-39 years, however, plural births are almost three times as frequent. The likelihood of a multiple birth also depends upon the race of the parents. The chances that a nonwhite mother will have twins are 13 in 1,000, or one fourth greater than they are for white mothers. Moreover, the disparity between the two groups is even larger for confinements yielding more than two babies. Thus, triplets are, relatively, 1¾ times as likely among nonwhite mothers as among the white, and quadruplets are almost four times as likely.—How Often Do Multiple Births Occur? *Statistical Bulletin Metropolitan Life Insurance Company*, April, 1954.

ADVANCES IN THE LABORATORY DIAGNOSIS OF POLIOMYELITIS

Thomas H. Weller, M.D., Boston

The establishment of a definitive diagnosis by laboratory procedures in an illness produced by an infectious agent is desirable. Whether or not such a diagnosis may be achieved is dependent on the availability of appropriate techniques. In this instance availability has reference not only to the existence of diagnostic methods but also to the adaptability of the specific techniques to usage in routine diagnostic laboratories. Thus, with reference to the problem at hand, the definitive diagnosis of poliomyelitis, it is to be noted that in a restricted sense laboratory methods for diagnosis have been available since 1909 when Landsteiner and Popper produced an illness associated with paralysis in monkeys on inoculation with material obtained from a patient with poliomyelitis. However, with the primate as the only experimental animal, diagnostic assistance could not be given the clinician dealing with a suspected case of poliomyelitis.

We will present here two developments in the field of poliomyelitis research as applied to the problem of diagnosis, both of which have minimized the role of the monkey. One development is the outgrowth of our observation, reported in 1949,[1] that the poliomyelitis viruses could be propagated in cultures prepared with human tissues. The other is the elaboration of techniques whereby representative strains of poliomyelitis virus have been adapted to rodents.[2] It will be apparent that these technical advances have increased the availability of diagnostic procedures in the general sense; yet it is to be emphasized that these methods are still undergoing evaluation and simplification. However, it appears that certain of the new techniques will prove adaptable to the requirements of the diagnostic laboratory and thus are of interest to the practicing physician. We will confine our discussion to those procedures designed specifically to identify an infection with poliomyelitis virus during life. We will not consider auxiliary laboratory tests (such as the spinal fluid examination) that do not yield a definitive diagnosis.

Whereas from the clinical standpoint poliomyelitis is justifiably considered a single disease, from the etiological standpoint it is necessary to conceive of this entity as produced by three related but immunologically distinct viruses. This concept, of fundamental import, was established by the collaborative efforts of the Committee on Typing of the National Foundation for Infantile Paralysis.[3] The laboratory diagnosis of poliomyelitis depends on the isolation from the patient of one of the three types of virus or else on the demonstration of immunologic phenomena indicating its presence. Virus isolation may be accomplished from the feces of the patient or, with less likelihood of success, from the pharyngeal secretions. Immunologic evidence consists in the demonstration of the appearance or increase of specific antibody in significant amounts upon examination of acute and convalescent phase blood specimens. The manner in which the new techniques may be applied to these ends will be considered.

TISSUE CULTURE PROCEDURES

The poliomyelitis viruses will multiply in vitro in a variety of cell types of human or of monkey origin. From the diagnostic standpoint, an important characteristic of these viruses in tissue culture is their cytopathogenicity or, in other words, their ability to destroy those cells in which they multiply. That this destructive effect is specific is manifest by the fact that cell destruction is prevented by homotypic antibody. In practice, employing suitable controls, the presence of poliomyelitis virus isolated in a culture may be recognized by direct microscopic observation of the cellular degenerative changes or else indirectly by recording reduction in metabolic activity of the affected cells. The virus may then be identified as to type by combining it with appropriate antisera and observing the presence or the absence of cytopathic activity upon inoculation of the mixtures into a second set of cultures. In a similar manner the neutralizing antibody content of blood from a patient may be assayed by determining the smallest amount of serum that will prevent cytopathic activity in vitro.

The development of these techniques has been reviewed elsewhere.[4] In brief, cultures of human or of monkey tissues are employed and are usually prepared by planting tissue fragments or cells on the wall of small test tubes that are incubated in a revolving drum or else in a stationary slanted position. This type of culture wherein cells are grown on glass or on a plasma substrate has proved a more sensitive indicator of virus than have flask or "suspended cell" cultures. The type of cell likewise affects the sensitivity of the system. When available, human kidney tissue has given the best results in our laboratory [5]; others are utilizing cultures of monkey kidney tissues as routine, and techniques for the preparation

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From the Research Division of Infectious Diseases and the Department of Medicine, the Children's Medical Center, Boston, and the Department of Tropical Public Health, Harvard School of Public Health.

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of standardized cell suspensions have been developed.[6] Syverton and Scherer [7] introduced the use of a stable strain of malignant human epithelial cells for in vitro poliomyelitis studies; they have demonstrated that these cells may be maintained in continuous culture in a central laboratory, and that subcultures may be distributed to diagnostic laboratories as needed. For virus isolation studies, either cultures of kidney cells or cultures of HeLa cells appear to yield optimal results; such epithelial cells appear to be particularly refractory to certain "cytotoxic" substances that may be encountered in human feces. If monkey tissues are utilized, as many as 2,000 cultures may be prepared from the kidneys of one animal.

IN VITRO ISOLATION

Although the first report of the in vitro isolation and identification of poliomyelitis virus appeared only three years ago,[8] the technique is now in general use in research laboratories. Fecal specimens or throat washings are prepared for inoculation by centrifugation and the addition of antibiotics to prevent bacterial and fungal growth. The efficiency of the procedure will be illustrated by a few representative results. In an extension of our studies, Kibrick and Enders [5] have isolated 136 strains of virus; a poliomyelitis virus was recovered from 98% of cases with paralytic disease. Syverton, Scherer, and Elwood [7] have recently reported the isolation of 300 strains of virus utilizing HeLa cultures. Melnick and co-workers,[6a] employing cultures of monkey kidney tissue, have recently described the isolation of 76 additional strains.

The results of viral isolation studies in cases bearing a clinical label of "nonparalytic" poliomyelitis are of interest. In a group of 84 such patients observed at the Children's Hospital, poliomyelitis virus was recovered from only 35, or 42%.[5] There are two obvious explanations for this comparatively low recovery rate from "nonparalytic" cases. First, the amount of virus excreted may bear some relationship to the severity of the clinical process; Ward and co-workers [9] have suggested that mild cases do excrete less virus. A second explanation is that certain cases in this clinical category represent not poliomyelitis but illnesses produced by other, as yet poorly defined, infectious agents. There is now ample evidence to support this view. In various laboratories, the application of the tissue-culture method has resulted in isolation of unidentified viruses from patients with so-called nonparalytic poliomyelitis; these have been termed by Melnick [10] "orphan viruses," for we know little of the diseases they produce. These viruses may be responsible in part for the syndrome of "aseptic meningitis."

IN VITRO DETECTION OF NEUTRALIZING ANTIBODY

Classically the diagnosis of a viral infection by serologic means has been based on the demonstration of neutralizing, complement-fixing, or hemagglutinin-inhibiting antibodies. Practical methods for the detection of the first two types of poliomyelitis antibody are now available, although their clinical usefulness is not yet fully defined. Inhibition of the in vitro cytopathic action of the poliomyelitis viruses provides one method of assay of neutralizing antibody. Such antibody usually appears early in the disease, and may even be well developed at the time the patient is admitted. To perform the test, appropriate serum dilutions are prepared and incubated with known amounts of virus; each virus-serum mixture is then inoculated into one or more cultures. The cultures are observed after an appropriate period for evidence of specific degenerative changes. High levels of the antibody for the responsible virus are a characteristic finding; on occasion there may be some increase in heterotypic antibody.[6a] This method is now being applied on a limited scale for diagnostic work. In our own hands, it has on occasion been useful. For example, in a young patient with thoracic herpes zoster and pleocytosis, there was questionable involvement of the right leg; demonstration of a marked rise in type 1 poliomyelitis antibody titer indicated a concurrent infection with poliomyelitis virus.

IN VIVO DEMONSTRATION OF NEUTRALIZING ANTIBODY

The recent adaptation of type 3 poliomyelitis virus to mice by Li and Habel [2b] and of type 1 virus to mice by Li and Schaeffer [2c] has made available rodent-adapted strains of all three types and has thus provided an alternative to tissue-culture methods for the assay of neutralizing antibody. Li and Schaeffer [11] have noted a close correlation between the results of mouse and of tissue-culture neutralization tests and have demonstrated that the mouse neutralization method may be utilized to establish age-antibody distribution curves for all three types of virus in a population group. Reports on applications of this new approach to the problem of diagnosis should shortly be forthcoming.

DEMONSTRATION OF COMPLEMENT-FIXING ANTIBODY

In 1952, Svedmyr, Enders, and Holloway [12] described a complement fixation test for poliomyelitis employing antigens derived from concentrated tissue-culture fluids. Although earlier a satisfactory antigen for type 2 virus had been prepared by Casals from infected mouse brain material, the development reported by Svedmyr made available antigens for all three types. Various workers have now confirmed this observation. Using strains of poliomyelitis virus that multiply in vitro in high titer (10^6 to 10^8 TC_{50} doses per milliliter, where TC_{50} = the dose in tissue cultures that induces infection in 50% of cultures), satisfactory tests have been obtained with crude

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6. (*a*) Melnick, J. L., and others: Poliomyelitis Viruses in Tissue Culture: 7. Experiences with Viral and Serological Diagnostic Procedures, Yale J. Biol. & Med., to be published. (*b*) Youngner, J. S.: Monolayer Tissue Cultures: 1. Preparation and Standardization of Suspensions of Trypsin-Dispersed Monkey Kidney Cells, Proc. Soc. Exper. Biol. & Med. **85**: 202 (Feb.) 1954.

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8. Robbins, F. C.; Enders, J. F.; Weller, T. H., and Florentino, G. L.: Studies on the Cultivation of Poliomyelitis Viruses in Tissue Culture: 5. Direct Isolation and Serologic Identification of Virus Strains in Tissue Culture from Patients with Nonparalytic and Paralytic Poliomyelitis, Am. J. Hyg. **54**: 286 (Sept.) 1951.

9. Ward, R.; LoGrippo, G. A.; Graef, I., and Earle, D. P., Jr.: Quantitative Studies on Excretion of Poliomyelitis Virus: A Comparison of Virus Concentration in the Stools of Paralytic and Non-Paralytic Patients, J. Clin. Invest. **33**: 354 (March) 1954.

10. Melnick, J. L.: Application of Tissue Culture Methods to Epidemiological Studies of Poliomyelitis, Am. J. Pub. Health **44**: 571 (May) 1954.

11. Li, C. P., and Schaeffer, M.: Serum Neutralization Tests in Mice and in Tissue Culture Against 3 Types of Poliomyelitis Virus, J. Immunol. **72**: 123 (Feb.) 1954.

12. Svedmyr, A.; Enders, J. F., and Holloway, A.: Complement Fixation with the 3 Types of Poliomyelitis Viruses Propagated in Tissue Culture, Am. J. Hyg. **57**: 60 (Jan.) 1953.

fluids as removed from infected cultures. Antigen production for large-scale diagnostic use is therefore feasible.

In the performance of the complement fixation test by Svedmyr and co-workers, not infrequently cases of poliomyelitis exhibited heterotypic as well as homotypic antibodies. These cross reactions are probably due to the presence of both type-specific and group-specific antigens in the poliomyelitis viruses. To reduce such cross reactions Black and Melnick [13] modified the test by the use of minimal quantities of complement and of antigen. In their study of 49 poliomyelitis cases yielding virus, a rise in homotypic antibody was observed to occur in 90% of the cases. Furthermore, homotypic complement-fixing antibody was rarely demonstrable before seven days after onset of the disease and often increased after the third week. In view of these findings it appears that some modification of the complement fixation test employing tissue-culture antigens will achieve general acceptance as a practical diagnostic procedure.

COMMENT

During the past five years a variety of new approaches to the problem of the laboratory diagnosis of poliomyelitis have been developed. These advances indicate that procedures applicable to the conditions of the diagnostic laboratory will be available in the future. At present, the clinical diagnosis of nonparalytic poliomyelitis might be termed one of "guilt by association." If paralytic poliomyelitis is prevalent in a community, the patient with signs of meningeal irritation and pleocytosis is usually labelled a "nonparalytic" case rather than a case of "aseptic meningitis of unknown cause." In such instances, it should shortly be feasible to evaluate the role of the poliomyelitis viruses with the assistance of factual rather than of circumstantial evidence.

300 Longwood Ave. (15).

13. Black, F. L., and Melnick, J. L.: Specificity of the Complement Fixation Test in Poliomyelitis, Yale J. Biol. & Med., **26**: 385 (April) 1954.

PRACTICAL AND THEORETICAL CONSIDERATIONS WITH REGARD TO VACCINATION IN POLIOMYELITIS

John R. Paul, M.D., New Haven, Conn.

Artificial immunization against poliomyelitis has been the target toward which research workers have been aiming for well over 40 years. During this period a number of attempts at active immunization in man have been tried in this country, notably in 1935, when several thousand children were inoculated with suspensions of infected monkey cord in which the virus of poliomyelitis had been inactivated by formalin or other chemicals. These tests were made during the poliomyelitis season, at a time when there was limited appreciation of the fact that there was more than one immunological type of poliomyelitis virus, and when methods for measuring the poliomyelitis antibody content of serum were crude and very expensive. For this and other reasons it is not surprising that this "early trial" was unsatisfactory, although several thousand children had been inoculated before the unsatisfactory features came to light.[1] The 1935 experiments were quickly terminated when certain accidents, which to this day have not been adequately explained, occurred, and this unfortunate circumstance seems to have set the clock back for at least 15 years in the progress of immunization against poliomyelitis.

From the Section of Preventive Medicine, Yale University School of Medicine.

Read in the Symposium on Recent Advances in Poliomyelitis before the Joint Meeting of the Section on Experimental Medicine and Therapeutics and the Section on Internal Medicine at the 103rd Annual Meeting of the American Medical Association, San Francisco, June 24, 1954.

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1. Vaughan, H. F.: Discussion of Poliomyelitis Papers, Am. J. Pub. Health **26**: 143, 1936.

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3. Hammon, W. M.; Coriell, L. L.; Wehrle, P. F., and Stokes, J.: Evaluation of Red Cross Gamma Globulin as a Prophylactic Agent for Poliomyelitis: 4. Final Report on Results Based on Clinical Diagnosis, J. A. M. A. **151**: 1272 (April 11) 1953. Hammon, W. M.: Limitations in the Use of Gamma Globulin in Poliomyelitis, Am. J. M. Sci. **226**: 125, 1953.

Today the goal before us is, as Sabin [2] has aptly said, to find a practical and safe process to produce artificially what nature does for 99 or 99.9% of the population, and to do this without incurring the 1 to 100 or 1 to 1,000 risk of paralysis that is the price paid for acquiring immunity to poliomyelitis naturally. I might point out here that the problem differs in different parts of the world and that there are several approaches to it, but the main ones are only two: passive immunization, by injecting specific antibodies; and active immunization, by injecting a substance that will induce specific antibodies in the vaccinated person.

PASSIVE IMMUNIZATION

Dr. W. McD. Hammon has said that a low level of circulating antibodies might be sufficient to confer some protection against paralytic poliomyelitis, and it was on this basis, together with regard for the protective effect of gamma globulin in measles and infectious hepatitis, that he and his collaborators carried out, in 1951 and 1952, a large-scale trial of gamma globulin as a prophylactic measure against poliomyelitis in more than 25,000 children during the epidemic seasons of those years. The results and the indications for use of gamma globulin have been given in detail in Hammon's previous paper,[3] and in his part of this symposium (see this issue, page 21). They recorded no serious accidents and very few bad reactions, and indicated that gamma globulin in the dosage used appeared to afford protection against the paralytic disease over a period of two to five weeks. On the basis of this 1951-1952 experience, and with the realization that gamma globulin was not a very efficient agent for the wholesale prophylaxis of poliomyelitis, the use of a rationed supply in specified situations was widespread in this country during the summer season of 1953. Two particular recommendations were

made: that it be used in household and intimate contacts of clinically diagnosed cases; and that it be used within epidemic areas where the risk to the juvenile population was high.[4] The results of this 1953 experience in this country were reviewed early this year by a National Advisory Committee whose report has been recently published.[5] The committee's decisions may not be the last word, particularly as it was pointed out in this report that the large-scale use of gamma globulin in 1953 resulted from a widespread public demand, and its administration was not done on an experimental basis. Nevertheless, it was decided that the administration of gamma globulin to familial associates with poliomyelitis had had no significant influence on the severity of paralysis or other features of the subsequent cases that developed in the exposed families. The results of the mass use of gamma globulin, in certain epidemic areas and populations where it might have been expected to be effective, suffer from the fact that most of the gamma globulin inoculations were given too late to be expected to have much effect, and the data from this trial were obtained in situations that lacked suitably chosen controls. In any event the committee could not say that the so-called mass use produced no effect. At best, therefore, gamma globulin can be regarded as having, as Hammon originally claimed, only limited usefulness as a prophylactic measure for the paralytic form of poliomyelitis. Its use will certainly not be abandoned.

ACTIVE IMMUNIZATION WITH KILLED VIRUS

Whereas with passive immunization the susceptible child can expect only temporary protection, considerable expectation for a longer period of immunity might be placed on active immunization against poliomyelitis through the use of vaccines. The trend of research here has been directed toward the development of two different types of vaccines: one containing inactivated or killed material obtained from each of the three antigenic types of viruses, and the other containing live but avirulent strains of virus. With the latter it might be possible to administer a virus that would produce a harmless though immunizing infection. As to the former type of vaccine, the killed material, to date experiments in animals and a significant number of tests in man have shown that such materials derived from all three types of poliomyelitis viruses have induced the formation of type-specific antibodies in both monkey and man, and, at least in the monkey, some postvaccinal resistance to infection with homologous challenged virus can be produced.[6] This has been a great step forward, carrying with it at long last the possibility that poliomyelitis vaccines may actually become available to the health officer and to practicing physicians in the not too distant future. But it is also clear that such experiments must now be placed on a quantitative rather than a qualitative basis in which the inoculation dose and the degree and duration of the antibody response, together with the fate of the person vaccinated as compared with suitable controls, will require careful measurement on a large-scale, long-term basis. Such an experiment as this is being carried out in the United States under the auspices of the National Foundation for Infantile Paralysis this year.

Before attempting to describe this experiment, it is important to list some of the developments that have come step by step in the long up-hill struggle to produce such an immunizing agent or agents, and have thus made "a human vaccine trial" possible. Among them has been the arduous task of separating the family of poliomyelitis viruses into three serotypes. Another is the development of knowledge of antibody patterns in various populations that has come to light recently. From a study of such antibody patterns one can deduce the "immunity status" of the population. These indicate that in some areas 95% of the local children reach "immunologic maturity," as far as all three types of poliomyelitis are concerned, by the age of 5 years; whereas in other places, such as this country, such "maturity" is not reached until the age of 15 or more. In the former population, vaccination of school children would not be indicated; in the latter, some type of artificial immunization is sorely needed.

But most important on the list of developments stands the demonstration in 1949, by Enders, Weller, and Robbins,[7] that poliomyelitis viruses not only multiply in nonnervous tissue cultures but also produce a characteristic cytopathogenic change by which the presence of virus can be readily recognized. This has eliminated in large measure the necessity of resorting to the expensive and complicated procedures involved in the use of monkeys, and has served as an invaluable source of virus propagated in a medium that presumably is not as dangerous an inoculum as is nervous tissue. Additional experimental observations have also made the outlook for artificial immunization against poliomyelitis in man more hopeful. Among them is the demonstration by Bodian [8] that the amount of antibody required to protect cynomolgus monkeys against paralytic infection experimentally acquired by the intramuscular or oral routes is much smaller than that needed to protect against infection by the direct intracerebral route.

On this basis, Salk and his associates, utilizing an extensive experience in their studies of formalinized vaccines against influenza, have turned to the inactivation of poliomyelitis virus by Formalin and have reported in detail the results of their first tests with "killed" tissue culture virus vaccines in human beings.[9] Much of the most recent work has not yet been published, but in one series of experiments [10] it appears that antibodies to all

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three immunologic types were induced by inoculation of small quantities of a trivalent vaccine. It has been clear that the postinoculation antibody response was far greater in those children who had preexisting type-specific antibody to one or more types than in those who were without preexisting antibody. It remains to be determined whether a course of inoculation covering one or more years will be advisable, and whether the use of adjuvants is indicated.

There are several requirements for an acceptable "killed" poliomyelitis virus vaccine. Obviously it must first of all be safe; its production must be practicable; and it should produce immunity that one might hope would be lasting. It should be remembered that the objective is not to vaccinate against poliomyelitis infections in general, but to vaccinate against paralytic poliomyelitis; a mild nonparalytic or inapparent infection can actually be a desirable event.

To meet the first criterion, the vaccine must not produce paralytic poliomyelitis. It must not be a potential source of another pathogenic agent, which might be more difficult to inactivate than poliomyelitis virus—as, for instance, hepatitis virus. Furthermore, as the "vaccine" may contain some foreign protein, and as multiple and spaced inoculations will undoubtedly be necessary, the material must not give rise to anaphylactic reactions or organ damage that might be brought to light only on repeated inoculation. This latter is more than a theoretical consideration, for reinoculation may be done not only after a few weeks, but after the passage of a year or more. There is, however, no longer a need to speculate on potential side-effects that may result from the use this year of Dr. Salk's vaccine. The stage is now set for direct observation, and the preliminary verbal reports to date indicate a remarkably low incidence of anything that could be termed an untoward effect.

It is all-important to determine whether the postvaccinal "immunity" of this type would be permanent or relatively short lived. In other words, the duration of artificial immunity induced by a "killed" virus vaccine might conceivably be weak and short; it might be enhanced from time to time artificially, by booster inoculations, or naturally, by "booster infections," if the person vaccinated were periodically exposed to poliomyelitis viruses; but as yet no one really knows how long such artificial immunity may persist. The point is that poliomyelitis is usually a more severe disease in adults than in young children, and it should be determined as soon as possible whether a course of active immunization with a "killed" virus would eliminate, or merely postpone, future infection. If the infection is merely postponed, then immunization might even have to be repeated periodically, conceivably for the rest of a person's life! On the other hand, it may be that a minimal amount of immunity produced by a weak immunizing agent might be sufficient to give the person vaccinated just enough resistance to modify his subsequent poliomyelitis infection. This would be desirable, provided the modifying effect was that of eliminating paralysis.

An important feature of any trial of such material is that it would be better not to carry it out during the poliomyelitis season, and particularly during epidemics. Observations within the past four years have indicated that inoculation with certain vaccines of various kinds during poliomyelitis epidemics may precipitate paralysis in an exposed person.

ACTIVE IMMUNIZATION WITH LIVE VIRUS

Another possible approach to the task of active immunization, and one that, if successful, would perhaps simplify the problem of vaccination and eliminate some of the theoretical dangers, is using not a killed vaccine but a live avirulent virus, duplicating the mechanism of natural infection and following the lead established long ago by Jenner in the prevention of smallpox, and later followed by Rockefeller Foundation workers in the prevention of yellow fever. The situation is triply complicated in poliomyelitis because strains of all three types of virus would have to be discovered or produced, each of which would have permanently lost its power to cause paralysis but would have retained its power to immunize.

The observations of Koprowski and his associates [11] have pointed the way here. His strain of type 2 poliomyelitis virus, which after passage through cotton rats lost much of its virulence on intracerebral injection in monkeys, produced antibodies in high titer after feeding a single dose to two groups of children. Cabasso and his associates [12] used the same principle in adapting another type 2 poliomyelitis strain to chick embryos. Another group of workers, Enders, Weller, and Robbins,[7] have reported that a type 1 strain of poliomyelitis virus after serial passage in tissue cultures lost most but not all of its capacity to produce paralysis in intracerebrally inoculated monkeys. Studies subsequently carried on in at least two other laboratories have confirmed the fact that this tissue-culture attenuation has promise in giving rise to variant strains that ultimately might be used for human vaccination. It would appear, however, that no series of completely avirulent strains of poliomyelitis virus is yet available for any large-scale trial in human beings.

It is heartening that enough information has been collected on this problem of active immunization against poliomyelitis to warrant its extensive trial on human subjects. But before making predictions a warning should be made; in spite of lay impressions gained from radio and newspaper publicity, there is no real or official "vaccine" against poliomyelitis yet. A number of substances are being prepared in several laboratories, one of which is being tried to see whether it is going to be worthy of the name of vaccine. The proof will not be at hand until hundreds of thousands of children have been inoculated and their progress followed to determine statistically over the years whether the age incidence of clinical or paralytic poliomyelitis is reduced in the groups of vaccinated subjects as compared with suitable control groups, and, if the incidence is reduced, for how long. The answer will probably not be obtained this year, but a hopeful beginning has been made.

333 Cedar St. (11).

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12. Cabasso, V. J., and others: Poliomyelitis: 3. Propagation of MEF1 Strain of Poliomyelitis Virus in Developing Chick Embryo by Allantoic Cavity Inoculation, Proc. Soc. Exper. Biol. & Med. **81**: 525, 1952.

EVALUATION OF RED CROSS GAMMA GLOBULIN AS A PROPHYLACTIC AGENT FOR POLIOMYELITIS

5. REANALYSIS OF RESULTS BASED ON LABORATORY-CONFIRMED CASES

William McD. Hammon, M.D., Dr.P.H., Pittsburgh, Lewis L. Coriell, M.D., Ph.D., Camden, N. J.
Ernest H. Ludwig, Ph.D., Pittsburgh, Robert M. McAllister, M.D., Camden, N. J.
Arthur E. Greene, Ph.D., Philadelphia, Gladys E. Sather, M.P.H., Pittsburgh
and
Paul F. Wehrle, M.D., Baltimore

In previous reports [1] we presented an analysis of the findings of a controlled field study [2] on the prevention of paralytic poliomyelitis by gamma globulin. The diagnosis of the cases included in these earlier reports was based entirely on rigid clinical criteria previously outlined,[2a] since no laboratory data were available at that time. The accuracy of such criteria has never been completely evaluated, because suitable laboratory tests have only recently become available, but errors in etiological diagnosis were to be expected even though only paralytic cases were included. In this paper we are presenting a reanalysis of results based exclusively on laboratory-confirmed cases. We shall also make reference to the United States Public Health Service reports [3] concerning the general use of gamma globulin in 1953 as they relate to data from our controlled study.

LABORATORY METHODS

Infection with poliomyelitis virus can be determined by either of two methods—isolation of virus or serologic response. All tests for virus isolation in this study were made on specimens of feces, usually a pool of three specimens collected from the subject on three consecutive or nearly consecutive days. These specimens were obtained as soon as possible after diagnosis. Tests were made in tissue cultures of human uterine cells or monkey kidney cells, usually the latter. When negative, the test was usually repeated after attempting to concentrate any virus present by ultracentrifugation. Monkeys were also often used for the second test. Viruses isolated were identified by tissue culture neutralization tests against type-specific immune monkey serums [4] prepared for each of the three recognized types of poliomyelitis virus. If the virus was not neutralized by one of these antiserums, it was tested against a pool of all three to eliminate the possibility that more than one type of virus was present in the person from whom the culture material had been obtained.

Serologic tests of two types, neutralization and complement fixation, were performed. All serums from one subject were always tested simultaneously, and all tests were quantitative in nature, beginning with a dilution of 1:4 and usually carried through 1:256. Neutralization tests were performed in roller tubes using suspensions of cells from trypsinized monkey kidneys. Two tubes were used for each dilution and each type of virus. Fourfold serum dilutions were used. A dilution of each type of virus representing 100 ID_{50} (infective dose for 50% of tubes inoculated) for tissue cultures was used. Complement fixation tests were carried out by the Svedmyr [5] modification of the Fulton and Dumball drop technique. Antigens were concentrated from fluid from tissue cultures [6] of each of the three types of virus by the method used by Svedmyr and co-workers, and the number of antigen units used was similar to that used by these workers.[5] Twofold serum dilutions were used throughout in the complement fixation tests.

INTERPRETATION

Isolation of poliomyelitis virus was accepted as diagnostic. In the absence of virus isolation, a fourfold or greater rise in the neutralization test was accepted unless complement fixing antibodies for that type of virus remained at a level below 1:4. A complement fixation change alone was accepted only when a neutralization test was not done and when at least a fourfold change in titer occurred for only one virus type. A series of serums remaining negative for complement fixing antibody was considered as strong evidence against current poliomyelitis infection. The interpretation of the complement fixation test will be discussed in a separate paper.

From the Department of Epidemiology and Microbiology, Graduate School of Public Health, University of Pittsburgh (Dr. Hammon, Professor and Head of Department, Drs. Ludwig and Wehrle, Miss Sather), Camden Municipal Hospital for Contagious Diseases and Department of Pediatrics, School of Medicine, University of Pennsylvania (Drs. Coriell and McAllister) and Research Department, Children's Hospital of Philadelphia (Dr. Greene).

Read in the Symposium on Recent Advances in Poliomyelitis before the Joint Meeting of the Section on Experimental Medicine and Therapeutics and the Section on Internal Medicine at the 103rd Annual Meeting of the American Medical Association, San Francisco, June 24, 1954.

Dr. Wehrle is now at the Poliomyelitis Research Center, School of Hygiene and Public Health, Johns Hopkins University.

This paper was discussed by Drs. Henry D. Brainerd and C. Henry Kempe, San Francisco.

This study was aided by a grant from the National Foundation for Infantile Paralysis, Inc.

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6. Crude virus suspension for concentration was obtained from Dr. William Wood, Connaught Medical Research Laboratories, University of Toronto, Canada.

TYPES OF VIRUS RESPONSIBLE FOR EPIDEMICS

Poliomyelitis viruses have been isolated from 245 of the patients and contacts in the epidemics included in the Utah, Texas, and Iowa studies of 1951 and 1952. Table 1 presents these isolations by area and by type of virus. It will be noted that all three types of poliomyelitis virus were represented in a proportion reasonably consistent with the recognized occurrence of these types in the United States in the last few years—89% type 1 and few of the other two types. In addition, untypable and as yet unidentified viruses were isolated in 16 instances.

TABLE 1.—*Virus Isolations from Stools in Three Epidemic Areas*

Area	No. of Patients Tested	Poliomyelitis Virus Type 1	Type 2	Type 3	Total No.	Total %	Other Virus Agents	Total Virus Isolations No.	%
Utah	96	26	3	0	29	30.2	2	31	32.2
Texas	136	66	13	12	91	66.9	11	102	75
Iowa-Nebraska	163	125	0	0	125	76.7	3	128	78.5
Total	395	217	16	12	245	62	16	261	66.1
Distribution of poliomyelitis viruses, %		88.6	6.5	4.9	100				

Patients in whom clinically diagnosed poliomyelitis developed four days through eight weeks after injection with gamma globulin or gelatin were found to be infected with the types and kinds of viruses shown in table 2. Comparative data on patients of the same age who did not receive injections are included. These data show that all proved cases of poliomyelitis developing in those receiving gamma globulin were due to type 1 virus, the type predominant in all areas. Cases of paralytic poliomyelitis that gamma globulin failed to prevent were not due to the types of poliomyelitis virus associated less often with clinical disease. Also, it can be observed that poliomyelitis virus was isolated less often from those patients who had received gamma globulin than from those who had not and that viruses not identified as poliomyelitis were isolated oftener from the former group. The greater frequency of isolation of poliomyelitis virus from uninoculated patients possibly reflects the under-reporting of the disease and consequent greater average severity of those cases that are reported, as pointed out in a previous report.[1b] This elimination of persons with mild illness from those brought to our attention would tend to decrease the number of diagnostic errors and thus increase the proportion of patients from whom virus could be isolated.

LABORATORY CONFIRMATION OF DIAGNOSES

Isolation of virus from feces, quantitative neutralization tests, and complement fixation tests on serial serum specimens, as described above, were used for confirmation of the presence of poliomyelitis. When, occasionally, a diagnosis could not be definitely confirmed on the basis of available specimens from the patient, virus isolation from one or more sibling contacts was accepted as adequate evidence of the cause of disease. A very serious endeavor had been made to obtain a series of suitable specimens both from each patient and from his sibling contacts, but this did not always meet with complete success. At the time of collection of specimens the type of inoculum received by the patient was unknown, so it is presumed that failures to obtain specimens are randomly distributed between patients receiving the two types of inoculum. Every case without adequate laboratory tests or specimens, except those in which the patient died or infection was proved in a sibling, has, therefore, been excluded from consideration. In the significant time interval (four days through eight weeks after injection) only three such cases were encountered, one in the gamma globulin group and two in the gelatin control group.

After careful review of the laboratory data, 10 cases formerly included among the gamma globulin group and 13 from the group given gelatin have now been omitted from this study. Those omitted will be considered briefly, except one in the former group and two in the latter that had their onset in extremely early or late time periods. As mentioned above, during the period of greatest importance in this study (four days through eight weeks after injection) one case was excluded from the gamma globulin group and two from the gelatin group because of the lack of suitable specimens for laboratory testing. There remain for consideration among the excluded cases in the significant time period eight in the gamma globulin group and nine in the gelatin group. A brief review of pertinent findings for these is presented in table 3. It will be noted that the severity was minimal (muscle score 5 or less, 0.9% total possible muscle involvement) in all the patients given gamma globulin and in five of those receiving gelatin. In the latter group one case was severe, two included moderate involvement, and one was mild. In the patient with the severest involvement (case 9) tests on feces yielded no virus; neutralization tests on three serum specimens between the 8th and 59th day after onset showed no antibody rises; and no change occurred in titers of these three serums to complement fixing antigens of the three poliomyelitis virus types. The patient in case 11, with moderate involvement, yielded a virus from the feces that was not typable as poliomyelitis; an untypable virus was also obtained from one sibling and no virus from two other siblings. Two serum specimens collected from this patient 6 and 23 days after onset showed no rises in neutralizing antibody titers but showed a decreasing titer by complement fixation with two types of poliomyelitis virus. We have encountered this apparent, nonspecific, or broad complement fixation response often in patients infected with viruses nontypable as poliomyelitis and without poliomyelitis-neutralizing antibody response, and it has occasionally been observed in patients from whom no virus was isolated. We therefore do not con-

TABLE 2.—*Virus Isolations from Patients with Clinically Diagnosed Poliomyelitis**

Inoculum		Poliomyelitis Virus Type 1	Type 2	Type 3	Total	Other Virus	No Virus Isolated	Total
Gamma globulin	No.	13	0	0	13	3	12	28
	%	46.5	0	0	46.5	10.7	42.8	100
Gelatin	No.	39	3	3	45	3	21	69
	%	56.5	4.4	4.4	65.3	4.4	30.4	100
None	No.	43	3	2	48	4	14	66
	%	65.2	4.6	3	72.8	6.1	21.2	100
Total, gelatin and not inoculated	No.	82	6	5	93	7	35	135
	%	60.7	4.44	3.7	68.8	5.18	25.9	100

* Poliomyelitis-like clinical syndrome involving the central nervous system with onset four days through eight weeks after inoculation with gamma globulin or gelatin, or during a similar interval after midclinic day if not inoculated.

sider a complement fixation change diagnostic unless the change is for only one type of virus. Two specimens of serum collected about one month apart from one sibling, age 3, of the patient in case 11 contained no neutralizing antibody to any of the three poliomyelitis virus types. No other serologic tests were performed on this group of siblings. In the other case of moderate severity, case 10, there was no detectable virus in the stool and none was obtained from four child siblings nor from the two parents. No neutralizing antibody increases were observed between two specimens of serum taken 4 and 29 days after onset, but by complement fixation test there was a fall in titer against two antigen types. Two blood serums collected from one sibling of the patient in case 10 showed a neutralizing antibody rise of group still lack significance, owing to small numbers. The likelihood that protection of a waning degree does persist through this period is increased by the fact that in each of these three weeks the difference is in the same direction. The cases occurring in the sixth through the eighth week by single weeks are: sixth week, one in the gamma globulin and three in the gelatin group; seventh week, one and four; and eighth week, one and two, respectively. Further indication of the effect of omitting unconfirmed cases is seen in the percentage of cases occurring from the fourth day after injection through the eighth week. Cases of poliomyelitis occurring in those who received gamma globulin now represent only 19.7% of the total in the two injected groups, while they represented 25.6% in the previous, less reliable data.

TABLE 3.—*Cases of Clinically Diagnosed Poliomyelitis Not Confirmed by Laboratory Studies, Occurring Four Days Through Eight Weeks After Injection of Gamma Globulin or Gelatin*

						Laboratory Studies					
Inoculum	Case No.	Age of Patient	Muscle Score [1]	Onset Interval, Days *	Cerebrospinal Fluid	Virus Isolated from Feces	Serum Specimens, No.	Serum Interval, Days †	Neutralization Test	Complement Fixation Test	Other Family Members
Gamma globulin	1	6	3.95	15	No test	0	3	3-64	No rise	No change	No serologic change: 1 sibling
	2	3	3	18	0	Other ‡	3	1-57	No rise	No change	Other virus, no neut. rise: 1 sibling
	3 §	8	2.5	47	+	0	3	— 3-58	No rise	No change	No virus, no serologic change: 3 siblings
	4	6	1	36	No test	0	3	7-60	No rise	No change	No virus, no serologic change: 1 sibling
	5	11	1	13	No test	Other	3	5-60	No rise	Rise, 3 types	No siblings
	6	1	0	44	No test	0	0	....	.....	.....	No siblings
	7	9	0	7	No test	0	3	2-60	No rise	No change	No siblings
	8	8	0	7	No test	0	3	8-60	No rise	No change	No siblings
Gelatin	9	4	158.4	46	+	0	3	8-59	No rise	No change	No siblings
	10	4	62.3	6	+	0	2	4-29	No rise	Fall, 2 types	No virus: 4 siblings, 2 parents; ± neut. rise, no CF antibody: 1 sibling
	11	5	38.75	12	+	Other	2	6-23	No rise	Fall, 2 types	No virus: 2 siblings; other virus: 1 sibling; no neut. antibody: 1 sibling
	12	3	13.5	6	+	0	3	2-63	No rise	No change	No virus, ± neut. change, no CF antibody: 1 sibling
	13	5	5	9	+	0	3	0-60	No rise	No change	No virus, no serologic change: 1 sibling
	14	4	4.75	20	No test	Other	3	4-70	± rise, type 1	0, type 1; no change, others	No siblings
	15	5	3.5	44	No test	0	3	11-59	No rise	No change	No siblings
	16	3	2.5	13	+	Other	3	4-64	No rise	Change, 3 types	No siblings
	17 §	5	0	42	+	0	3	2-64	No rise	Change, 3 types	No virus, no serologic change: 3 siblings

* Interval in days from clinic injection to onset of illness.
† Interval in days from onset to collection of first and last specimens.
‡ Virus not typable as one of the three poliomyelitis virus types.
§ The patients in cases 3 and 17 were siblings, one of whom received gelatin and the other gamma globulin. The onset in case 3 was three days after the patient's blood had been examined as a contact of the patient in case 17. No virus was isolated from that blood specimen.

questionable significance to one type of virus but no detectable complement fixation antibody in either specimen for any of the three virus types. Based on such findings, this group of cases was excluded as poliomyelitis virus infections.

ANALYSES BASED ON LABORATORY-CONFIRMED CASES

In table 4 data on laboratory-confirmed, paralytic cases of poliomyelitis are compared with the results based on clinical diagnoses that were previously published. Several changes can be noted. Some protection during the first week (latter part of the incubation period) now appears more probable; protection during the next four weeks, highly significant before, is even greater; and protection in the period six through eight weeks after injection of antibody also appears greater, although the data on this time interval as an isolated

POSSIBLE CAUSE OF EXCLUDED CASES

The total exclusions made in the gamma globulin group in the four-days-through-eight-weeks period represent an elimination of 9 out of 22, or 41%, of cases diagnosed on a clinical basis as poliomyelitis, and in the gelatin group 11 out of 64, or 17.2%. Despite this proportional difference, an approximately equal number (9 and 11, respectively) was eliminated from each group. All these findings suggest that agents other than the recognized poliomyelitis viruses acted in these areas to produce a clinical syndrome that generally resembled mild or, possibly, even severe paralytic poliomyelitis. Such cases do not appear to have been prevented by gamma globulin.

Seven unidentified viral agents were isolated from the patients whose cases were excluded and their siblings (table 3). These have been tested against all types and

combinations of poliomyelitis antiserum as well as against gamma globulin in dilutions above those toxic to tissue cultures. No protective effect could be demonstrated against any. From uninoculated children who had laboratory-unconfirmed but clinically diagnosed paralytic cases and from patients from all groups, who had clinically diagnosed, nonparalytic cases that were not confirmed by laboratory tests, eight similar viruses not typable as poliomyelitis were isolated. Thus, 15 out of the total 16 unidentified strains of virus found in the whole study were isolated from patients with laboratory-unconfirmed but clinically diagnosed cases. The remaining unidentified virus was isolated from a patient with type 1 poliomyelitis infection that was adequately confirmed by serologic tests. These findings certainly suggest that one or more agents other than the recognized poliomyelitis viruses were active in these areas, producing a syndrome resembling paralytic and nonparalytic poliomyelitis. These agents were not significantly affected by gamma globulin in the amount used.

TABLE 4.—*Cases of Paralytic Poliomyelitis by Type of Inoculum and by Time of Onset After Inoculation*

	Laboratory-Confirmed Cases				Clinically Diagnosed Cases		
			Gamma Globulin			Gamma Globulin	
Interval After Inoculation	Total	Gelatin, No.	No.	%	Gelatin, No.	No.	%
1-3 days	7	4	3	42.8	4	4	50
4-7 days	16	10	6	37.5	12	8	40
2nd week	22	20	2		24	3	
3rd week	8	7	1		8	3	
4th week	5	4	1	10.5	4	1	15.2
5th week	3	3	0		3	0	
6-8 weeks	12	9	3	25	13	7	35
9-13 weeks	8	3	5	62.5	5	5	50
Total	81	60	21	25.9	73	31	29.8
Total incidence, 4 days-8 weeks	66	53	13	19.7	64	22	25.6

How many of the agents isolated may be identical has not been determined. Those tested to date do not have the properties associated with the Coxsackie viruses, but one of two tested in monkeys produced distinct pathological changes, observed in sections of the spinal cord and nerve roots taken at 9 and 30 days after injection.

USE OF GAMMA GLOBULIN

Prophylaxis.—A number of references have been made by others to the proportion of persons offered protection by gamma globulin. We have hesitated to make such a statement until more accurate diagnoses could be made, and we feel strongly that any such figure that is presented should be accompanied by a standard error, so that a concept of the confidence range is also presented. Unfortunately, no such confidence level can be calculated with any accuracy using the data available, since we cannot determine the size of the susceptible, exposed population involved in the experiment. This group was very large, however, certainly including several thousand children, so the figures have reasonable stability. We can only say now that in the experimental group, (1) with the potency and dose of antibody used, (2) when infection was due to poliomyelitis virus and not another agent or agents producing a similar clinical syndrome, and (3) when infection was acquired at such a time that the onset of disease occurred more than three days after receiving gamma globulin and not more than eight weeks after, 75.5% of those treated appear to have been protected from the development of paralytic disease. When the onset of disease occurred from one week through the fifth week after injection of gamma globulin and all other factors were unchanged, then 88% appear to have been protected.

If exposure and infection could be planned and timed to occur at will, gamma globulin would represent a reasonable agent for prophylaxis. As we have pointed out before,[7] however, the time of infection is so unpredictable, the immune status of individual persons in the population so completely unknown, and the ratio of children needing additional resistance to avoid the development of clinical disease so small (1:100 or 1:1,000) that the use of this agent would not ordinarily have been considered if anything else had been available. If the disease were other than poliomyelitis, which has such great emotional association, gamma globulin would certainly never be offered for any widespread use. This does not mean that gamma globulin prophylaxis has no practical application in poliomyelitis. If further research studies with suitable controls could be carried out the most advantageous application could probably be determined. Without such studies, application is largely done by guessing, with sharp differences of individual opinion.

Modification.—One of the most debated fields of application depends to some degree on the effectiveness of the agent in modifying disease when it fails to protect completely. Also of particular importance is its modifying or preventive action when it is given during the week before onset of disease. A new analysis has been made of data related to these problems, based only on the laboratory-confirmed cases (table 5). Unfortunately for analysis, the number of confirmed cases occurring in the group that received gamma globulin is very small; thus, significance of differences can be attained only if the differences are very large. In this analysis nonparalytic cases, confirmed by virology laboratory tests, are included for the first time. They had been excluded from the analyses previously reported because of the large proportion of diagnostic errors expected. These laboratory-confirmed, nonparalytic cases now are placed at the lower end of the severity scale and the fatal cases at the other extreme. This method of grading paralysis was originally developed for this study with the assistance of Dr. Jessie Wright [2b] and simplified slightly for application in the 1953 studies of the Public Health Service.[3] All scores are based on the final, 60 day muscle examination. This examination was the last of a series of three complete muscle gradings and many less complete examinations that were all made and recorded by members of the research team. Those included as paralytic, with 0 muscle score, had been observed to have significant paralysis at the time of one or more earlier examinations. The score can be transformed to a percentage muscle involvement by dividing by 560, the highest possible score. The muscle scores were arbi-

7. Hammon, W. McD.: Passive Immunization Against Poliomyelitis with Especial Consideration of the Effectiveness of Gamma Globulin, Bull. New York Acad. Med. **29**: 930-942 (Dec.) 1953; Limitations in the Use of Gamma Globulin in Poliomyelitis, Am. J. M. Sc. **226**: 125-130 (Aug.) 1953. Hammon.[1b]

trarily grouped into four broad classifications of severity for comparative purposes in table 5; because numbers are small, these are regrouped into two for calculating percentages. The first group contains the mildest cases, from nonparalytic cases to those with muscle scores up to 19 (3.4% total involvement), and the second group includes all severer cases, including the fatal cases. It will be noted that cases occurring during the first through the sixth week after inoculation were milder among those given gamma globulin than among those given gelatin. The difference observed for this period of time is significant.

Although the number of cases occurring during the first week is quite small, the direction of observed difference suggests that, as in measles and probably hepatitis, if prophylaxis in the late incubation period fails to prevent, it may modify. If one eliminates all cases with no residual paralysis at the time of the 60 day examination (this had to be done in the United States Public Health Service study in 1953,[3] since no reliable examinations were included in that study to detect paralysis at any earlier time), modification is obscured almost completely but protection is more obvious. In the total time period covered by the study 8 cases without residual paralysis may thus be eliminated from the gamma globulin group and 10 from the gelatin group. Six and one, respectively, are from the first week. Eliminating these cases without residual paralysis leaves 5 cases in the gamma globulin group and 13 in the gelatin group with onset in the first week after injection. This difference in incidence of disease with residual paralysis, though proportionately large (61% reduction) and important if confirmed, cannot be considered significant, however, since the number of observations is small (P=0.07). If confirmed, this difference would represent protection against residual paralytic poliomyelitis by gamma globulin given during the last week of the incubation period. If the cases without residual paralysis occurring during the following five weeks are discarded, the remaining differences of severity still suggest that modification may occur if prevention fails.

TABLE 5.—*Severity Distribution of Laboratory-Confirmed Cases of Poliomyelitis by Type of Inoculum and by Time of Onset After Inoculation*

Time Interval		Gamma Globulin					Gelatin				
		N.P.-0*	1-19	20-99	100+	Total	N.P.-0	1-19	20-99	100+	Total
1st week	No.	6	4	1	0	11	1	10	2	1	14
	%	91		9			78.6		21.4		
2nd-6th week	No.	2	2	2	1	7	7	5	17	9	38
	%	57.2		42.8			31.6		68.4		
7th-13th week	No.	0	2	2	3	7	2	2	5	1	10
	%	28.6		71.4			40		60		
Total	No.	8	8	5	4	25	10	17	24	11	62
	%	64		36			43.5		56.5		
Total, 1st-6th week	No.	8	6	3	1	18	8	15	19	10	52
	%	77.8		22.2			44.2		55.8		

* N.P. = nonparalytic. All numbers represent muscle scores. Fatal cases are included in the 100+ group.

Family Contacts.—The application of this information to use in family contact groups is obvious. Many statements have been quoted from authoritative sources by the lay press and have appeared repeatedly in health education publications to the effect that gamma globulin offers no protection if given after infection, i. e., during any phase of the incubation period. It has been stated that in the controlled experiments an effect was demonstrated only when gamma globulin was given before infection occurred. For this reason, it is explained, gamma globulin can be of no use when administered to family contacts, since most of these persons are exposed and in the incubation period before injection. Data in our previous reports, the only experimental or controlled observations on this problem to date, gave no such information. The more accurate data based only on laboratory-confirmed cases lend support to the hypothesis that antibody given during the incubation period has a beneficial effect that can be interpreted either as modification or as prevention, depending on the type of examination and threshold used to determine the existence of muscle weakness. Therefore, unless better evidence has become available, use of gamma globulin after recognized exposure among family contacts or any other contacts of known cases is supported by suggestive, though admittedly inconclusive, experimental data. At least, there is no basis for concluding that gamma globulin will not protect under such conditions.

In this reanalysis of experimental evidence based on laboratory-confirmed cases, a number of rather important differences appear between these data and impressions gained by many from the series of United States Public Health Service reports on general usage of gamma globulin in 1953. Since the complete report has finally been published and one of the authors of this paper was a member of the committee asked to revise and endorse the report, comment might be expected. The reader is referred principally to the original data[3] to form his own conclusions. All the reasons why controls are necessary in order to draw any valid conclusions that we stated in our description of planning for the trials of 1951 and 1952 were adequately demonstrated in the lack of controls in the 1953 study. Conclusions from mass prophylaxis were that no effect could be demonstrated. This is not surprising, since there was no constant value with which to compare the observed results. It was carefully stated in the report, however, that there was also no evidence to show

that gamma globulin did not have an effect. These not-too-revealing results had been anticipated by the committee before the study was made.

The report on family contact use, although attempting to present comparative data, in our opinion lacked such data. The inconclusive results obtained have been interpreted by many as conclusive. Comparative rates on which conclusions might be based would require data on the number of cases occurring among a known number of family contacts who did not receive gamma globulin and among a known number of family contacts who did receive gamma globulin, ages and other factors in the two groups being reasonably similar. Rates could then be compared for families in which the agent was and was not used. This was not done. Although the number of reported cases in each group was available, the two exposed populations from which the cases were derived were completely unknown; thus no rates for comparison could be calculated. Instead, a review of past experience in epidemic areas dating back to 1905 in Sweden and 1916 in New York, when age distributions and immunity status of persons in a family were entirely different from those found today, and of more recent experience that was almost entirely from very large urban epidemics was made, and from this it was pointed out that from 4.6% to 17.4% of all subsequent cases could be expected to occur 13 to 30 days after the onset of a family index case. This range for the normal expectancy would permit a 278% increase from the lower figure or a 74% reduction from the upper. The report reads, "With such variability it was evident that the possible preventive effect of gamma globulin would be observable in this type of study only if: (1) a very large series were studied; (2) gamma globulin were given very consistently to most family contacts; and (3) gamma globulin possessed a marked preventive effect." The series of cases was reduced by one type of elimination after another to a total of 415 cases. The patients in 257 (62%) of these did not receive gamma globulin at all or received it on the day of or the day after onset of poliomyelitis, when it would not be expected to have any effect. However, because there was no way to allocate cases by family or to separate these families completely, the 62% of essentially uninjected cases had to be included with the 38% in which the patients received the injection to determine what effect the agent had had. A total of only 30 cases occurred 13 to 30 days after the onset of the index case, and the patients in 11 of these had not been inoculated. This small, mixed group, then, was the crucial one for comparison with past experience when gamma globulin had not been used at all. These 30 cases, occurring in both injected and uninjected patients, were 6.6% of the total number of cases occurring after an index case. This percentage fell within the wide limits of 4.6% to 17.4% considered normal. It will be observed that two of three essential criteria for a successful study that had been set down in advance had not been fulfilled—"a very large series" and "gamma globulin . . . given very consistently to most family contacts." The report draws the following conclusions from this aspect of the study: "Thus the availability in 1953 of gamma globulin on a national basis for administration to household associates did not induce a discernible deviation in the classical epidemiological pattern of familial aggregation of the disease." This has been interpreted in many statements elsewhere as proof that incidence of disease was not affected by gamma globulin usage in family contacts.

Further highly suggestive evidence of the effectiveness of gamma globulin is presented in a report of its use in a number of epidemic areas in Mexico during the summer of 1953.[8] In these instances the areas selected, the time of starting inoculations, and the age groups given gamma globulin were more satisfactory than in most of the United States experience of the same year. The results support the effectiveness of use of the agent in suitable circumstances in mass immunization and also suggest its effectiveness in family contacts. Evidence for both is far from adequate but tends to confirm the earlier, controlled tests.

We prefer to base conclusions about the effectiveness of gamma globulin in any application on experimental observations with suitable controls, rather than on the type of data available from the 1953 studies described. There appears to be definite evidence from the controlled studies that gamma globulin protects when given under suitable circumstances. It is regrettable that these circumstances cannot be more completely described, but, the experiment was designed to answer one basic question: "Will this amount of antibody prevent poliomyelitis under any conditions of natural exposure?" and not, "How can gamma globulin be used most effectively in controlling the disease?" No experiment has been performed yet to answer this second question. The experimental evidence suggests, nevertheless, that protection derived from the amount of antibody given in the field tests began to have a noticeable effect late in the incubation period, was most effective when given at about the time of exposure or one week before, and continued with slowly diminishing effect for six to eight weeks. This information has some use in planning how to use the agent in disease prevention.

EFFECT OF GAMMA GLOBULIN ON INAPPARENT INFECTION

There has been considerable interest of a practical nature in the possible effect of gamma globulin on the incidence of inapparent infection and the development of immunity. To obtain information about this has required a great deal of laboratory work. The results of this phase of the study will be presented in detail in subsequent reports. No significant difference in the proportion of persons excreting virus can be detected among persons of the same age group who were contacts of patients with confirmed poliomyelitis, whether these contacts had received gamma globulin, gelatin, or no injection prior to infection. The same holds true for the proportion of significant rises in neutralizing antibody titers in a series of blood specimens collected from such contacts during an appropriate time period. However, when all aspects of the laboratory data are studied, including results of complement fixation tests, the height of titers to both serologic tests, the time required to reach high titers,

8. Calderon, C., and others: Results Obtained with Gamma Globulin in the Prevention of Poliomyelitis in Mexico, read before the United States-Mexico Border Public Health Association 12th Annual Meeting, Albuquerque, N. Mex., April 7-9, 1954.

the final percentage of infections confirmed as poliomyelitis by combining the results of all tests, and the direction of small differences below the order of significance, one cannot state with assurance that this level of injected antibody has shown no effect on infection. A slight interference has probably occurred, but the degree is negligible with administration of this amount of gamma globulin. This does not mean, however, that higher levels of antibody would be ineffective. We are inclined to interpret the slight effect observed at such an extremely low level as suggesting that a more important effect will be observed in persons with higher antibody levels, whether these are acquired from larger doses of antibody passively transferred or produced by vaccination.

SUMMARY

Appropriate specimens from patients with clinically diagnosed cases of poliomyelitis from the controlled field tests of gamma globulin made in Utah, Texas, and Iowa and Nebraska in 1951 and 1952 have been tested in the virology laboratory to determine which cases were or were not caused by one of the recognized types of poliomyelitis virus. A new analysis of the data on such confirmed cases was done to determine the effectiveness of gamma globulin in the prevention of poliomyelitis. Infections with all three types of poliomyelitis virus occurred in the area under study. Clinically diagnosed cases developing in those who received gamma globulin were due either to type 1 poliomyelitis or to agents other than poliomyelitis virus. During the period of time when gamma globulin appears to have shown some effect in preventing paralytic disease, an approximately equal number of cases in the groups receiving gamma globulin and gelatin could not be confirmed as poliomyelitis by laboratory tests. These unconfirmed cases represented 41% of the gamma globulin group and 17% of the gelatin control group. Viral agents isolated from these excluded cases were unaffected by type-specific poliomyelitis antiserums, a mixture of these serums, or gamma globulin. Of 16 such agents isolated, 15 came from patients whose disease could not be shown to be due to poliomyelitis virus or from the sibling contacts of those patients. The exclusion of disease not attributable to poliomyelitis virus from the study shows gamma globulin to be slightly more effective against poliomyelitis than it appeared formerly. Protection and/or modification to the point of eliminating detectable paralytic involvement by the time of the 60 day muscle examination probably occurred even in those receiving gamma globulin during the week before onset; protection was most conspicuous during the next four weeks after injection and appears to have lasted well into the period of six through eight weeks after injection. Protection appears to have been a combination of prevention and modification of disease. These data offer very suggestive, although not conclusive, evidence in support of the use of gamma globulin after exposure has occurred. Laboratory tests on contacts of patients with confirmed cases of poliomyelitis indicate that the amount of antibody given the patients in this study did not interfere with infection or the development of active immunity to an important degree.

THE ANESTHESIOLOGIST, THE POLIOMYELITIS TEAM, AND THE RESPIRATOR

William A. O'Brien III, M.D., Arthur E. Scott, M.D., Robert C. Crosby, M.D.
and
William E. Simpson Jr., M.D., Reno, Nev.

The practicing anesthesiologist is not a research scientist in the strict sense, yet by nature of his training and daily experience he qualifies as a clinical cardiorespiratory physiologist. He is alert to problems in vital medicine relating to deficiencies of pulmonary ventilation and inadequacies of the oxygen transport system, having faced such problems on many occasions. If he has been diligent in his work, has established good relations with the physicians of his area, and has earned their faith in his judgment and respect for his capabilities, he then finds opportunities extended for even broader service to the profession and to his community.

In what manner can the anesthesiologist render a greater professional contribution to his community than in furnishing the leadership necessary to the organization and continued function of a poliomyelitis team? This question came forcibly to us when at the beginning of a recent poliomyelitis season there were two near fatalities from bulbospinal poliomyelitis in our hospital. Indecision in the early management of these cases impressed us with the need for medical teamwork if future jeopardy was to be minimized. Consequently, a meeting of hospital and professional personnel was called for the express purpose of forming a poliomyelitis team.

PLANNING THE TEAM

Because of special training in the realm of cardiorespiratory physiology, the anesthesiologist was unanimously approved for primary responsibility in the care of respirator cases. It was established that he would be notified of all poliomyelitis patients entering the hospital, and that he would make the decision as to the necessity for tracheotomy and/or respirator care. Such decisions would be made after careful and thorough examination of each individual case, close harmony and consultation being maintained between the anesthesiologist and the pediatrician, internist, or general practitioner, as the case might be. Care while in the respirator was planned on a similarly cooperative basis, liaison being maintained between the various physicians on a

From the Department of Anesthesiology, Washoe Medical Center.
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given case by carefully recorded progress notes and repeated consultation either in person or by telephone. An immediate meeting of local nurses was called, and the facts were placed squarely before them concerning the vital need for nurses trained in the care of all types of poliomyelitis. Emphasis was placed on the fact that without the best nursing care life is impossible for a patient with a tracheotomy or a patient in a respirator. Volunteers for poliomyelitis nursing were given on-the-job training. In addition, classes were held for groups of three to six nurses, the one patient in a respirator then in the hospital being used for quiet but effective demonstration. Physicians also were in need of a review of poliomyelitis in its many aspects. To fill this gap several physicians, including the anesthesiologist, made trips to large centers in order to observe treatment methods. All possible information was gathered from the chief of staff, assistants, residents, interns, nurses, physical therapists, and engineers. The accumulation of ideas from all sources was then systematically pooled into the working knowledge of the poliomyelitis team.

Already the observation had been made that throughout the country death rates were uniformly high in the group of patients exhibiting severe respiratory or bulbar involvement. This was found to be true even where good physicians, good nurses, and good respirators were available. A prominent difference was noted, however, in the mortality reports from certain large centers [1a]; close study revealed that they had not only intensified certain therapeutic measures ("elective" tracheotomy, early respirator care, and careful daily laboratory control of blood chemistries), but that they had also developed special equipment of material aid in evaluating and treating patients with respiratory involvement. Two devices were most notable: (1) a special ventilation meter adapted to use on communicable disease wards and (2) an intermittent positive pressure respirator attachment.[1b] The logic behind the entire program was of such soundness and the mortality figures, as compared with other centers, so low as to convince us that we should emulate their methods. Equipment needs were rapidly satisfied, thereby bringing to an end the preparedness phase of our program. Yet to come was the compilation of proof that physicians can band together in small communities and save lives that would otherwise be lost without concerted effort.

TRANSPORT EXPERIENCES

Much has been written about the home treatment of the poliomyelitis patient, and a certain merit is to be found in these papers.[2] Likewise, some authors believe that transport in itself may act adversely on the disease and produce further involvement.[3] We criticize neither of these approaches; however, there is an apparent difference between applying them in a large community and applying them in a broad geographical area, such as ours, that is characterized by broad expanses of desert, great distances between small towns, and the rugged mountainous territory of the Sierra Nevadas. There are many regions of our country that have similar population and physical features. To them our experiences with patient transport may be of value.

It is not uncommon for us to receive cases several hundred air miles from Reno. Indeed, the anesthesiologist has on two occasions made flights to towns 300 miles distant in order to bring in a child with bulbospinal disease. Assistance to respiration on the return flight was afforded by employing a standard anesthesia machine carried in the plane. No pulse or respiratory changes were noted en route. Each child had a tracheotomy and was in a respirator within seven hours of the first call concerning him. The case of a 37-year-old man from an adjoining state vividly illustrates the possible danger in refusing to hospitalize an acutely ill patient because he does not exhibit respiratory difficulty. This patient was admitted to our hospital after two refusals from hospitals near his home. Within 12 hours after his admission vital capacity readings had fallen from normal levels to 1,200 cc. The patient required 43 days in the respirator and ultimately made a complete recovery.

We have had 41 seriously ill patients who have been transported from 33 to 300 miles by air, ambulance, or automobile. Their ages have ranged from 7 weeks to 59 years. Although impressions may be fallacious, we nonetheless feel that no patient suffered ill effects from transport. When patients were found to be in poor condition upon arrival at the hospital the invariable observation could be made that too great a delay had been allowed before the decision to transport was made. It would, therefore, appear more rational to transport patients to equipment centers early so that the shift from careful observation to definitive action can be quickly made. Any patient not requiring hospitalization can readily be sent home; however, patients requiring the respirator cannot always be transported fast enough.

VENTILATION STUDIES

A further observation we feel important is that we have repeatedly received patients who have been sent in only after it has become clearly obvious that they are getting into severe respiratory difficulty. A careful history has again and again revealed that the difficulty had been in progress several days before reaching an alarming state. Performing tidal air and vital capacity studies serially as a routine would have resulted in an earlier sounding of the alarm and earlier transport to or employment of respirator facilities. The use of the respirator must not await the development of cyanosis and respiratory emergency [4]; it must be employed early in any patient demonstrated to have a falling vital capacity. In no other way can the nervous system of a patient already damaged by the poliomyelitis virus be protected from the superimposed and equally damaging effects of asphyxia.[5] It is imperative, then, to measure the patient's breathing capacity. It does not matter how, but measure the breathing capacity.

1. (*a*) West, H. E., and Bower, A. G.: Treatment of Poliomyelitis Involving the Respiratory System, Am. J. M. Sc. **217**: 252-255 (March) 1949. (*b*) Bower, A. G.; Bennett, V. R.; Dillon, J. B., and Axelrod, B.: Investigation on the Care and Treatment of Poliomyelitis Patients, Ann. West. Med. & Surg. **4**: 561-582 (Oct.) 1950.

2. Batson, R.: Medical Management of Poliomyelitis Problem, Part 2, Am. Practitioner **4**: 305-311 (May) 1953.

3. Stimson, P. M.: Home Care for Acute Poliomyelitis, Postgrad. Med. **13**: 156-158 (Feb.) 1953.

4. McLoughlin, C. J.: Respirators, and How to Use Them, Arch. Phys. Therapy **23**: 336-340 (June) 1942.

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We have noted that some adult patients with adequate hemoglobin levels will tolerate a diminution of vital capacity to one-sixth of the calculated normal without showing clinical cyanosis. With vital capacities 60% of the calculated normal, however, the patient complains markedly of being "just tired" and sleeps very poorly, if at all. A number of these patients have never shown specific paralysis, only generalized weakness and a feeling of extreme fatigue. It has been interesting to note that after 12 to 15 hours in the respirator a significant rise of 500 cc. or more in vital capacity has occurred. One or two hours out of the respirator has uniformly, at this point, resulted in fatigue and a fall of vital capacity to previous levels.

RESPIRATOR EXPERIENCES

As pointed out previously, we have employed actual breathing measurements in serial fashion as one major guide to the institution of respiratory assistance. Obviously, a diminished vital capacity of any degree in a patient who is acutely ill, febrile, and in the throes of a still developing disease is of far greater immediate significance than a similar diminution of capacity in a patient who is already past the acute stage, is fever free, and is not faced with the possibility of advancing paralysis and further diminution in the ability to breathe. We have set no arbitrary ventilation levels as determinants for beginning respiratory assistance but have chosen instead to judge each individual case on the basis of the over-all clinical picture; however, we have found no patient in the acute phase who tolerated a sustained decrease in vital capacity below 60% of the calculated normal. Other important factors we consider during the unstable period are: (1) degree of fatigue generally as well as after deep breathing, (2) breath-holding ability, (3) use of accessory muscles of respiration, (4) degree of intercostal weakness as measured by manual restraint over lower rib cage, (5) type of breathing—thoracic or diaphragmatic, (6) asymmetry of chest expansion, (7) clinical determination of diaphragmatic descent bilaterally, (8) stethoscopic evidence of amount of air flow into the two lungs, (9) deltoid weakness or paralysis suggesting impaired diaphragmatic activity, (10) ability to rest or sleep, (11) apprehension, and (12) cyanosis. Certainly there is no substitute for constant watching of any patient who may require mechanized breathing assistance. Check and recheck; evaluate and reevaluate is a necessity. Once the important decision has been made to employ a respirator, great care is exercised in softening the psychological shock the patient will experience. The function of the respirator is explained, and stress is placed on the fact that it is nothing more than a breathing helper that can be adjusted to suit the patient. Other patients sleeping or lying quietly in respirators are pointed out.

Cycling of respirator pressure and rate is achieved through diligent consultation with the patient as to whether the tank breathes too much or too little, too fast or too slow. Arbitrary rates and pressures are used only as starting points. A satisfactory starting point we have employed, except in infants and children below five years of age, has been to place the mask of the ventilation meter over the patient's face and set the negative tank pressure at a level that will reproduce the patient's calculated tidal air. When respirator care is first instituted a good many patients have been found to require an artificially induced tidal exchange of approximately double their normal; however, we start with a reproduction of the normal and increase to the point of patient satisfaction, coupled, of course, with evidence of adequate clinical improvement and cessation of voluntary breathing out of cycle with the respirator. Later we attempt to decrease the artificial ventilation gradually.

High negative pressures may be required to produce adequate ventilation in large or obese adults. A 40-year-old woman, height 5 ft. 7 in. (170.18 cm.) and weight 220 lb. (99.8 kg.), required a negative pressure of 25 in order to provide a tidal exchange of 600 cc. The weight of her large pendulous breasts as a factor in immobilizing the chest must be given due consideration. Respirator cycling of infants is an exacting, painstaking, and difficult task. Experience has been limited in our series to one baby seven weeks old with tracheotomy and one baby two and one-half months old without tracheotomy. A juvenile respirator is needed if the rapid respiratory rate of an acutely ill infant is to be reproduced. In addition to the usual turning of patients to minimize the chance of atelectasis, we have employed several other techniques: (1) hyperventilating the patient several times daily by doubling the negative tank pressure for five minutes, placing the mask of a standard anesthesia machine over the patient's face and manually forcing oxygen or helium-oxygen into his lungs by compressing the breathing bag, and increasing the positive tracheal pressure by means of the respirator attachment in cases with tracheotomy, and (2) teaching a patient to close his glottis at the end of inspiration and attempt to cough. If the respirator is run to its maximum negative pressure and the large "bed pan door" opened suddenly at the end of inspiration a fairly effective cough may be simulated. The negative respirator pressure may be augmented further (–40 to –50) by the simple expedient of taking a tank-type vacuum cleaner and sealing the hose in a respirator port. This additional pressure will greatly boost the negative respirator pressure.[6]

Prolonged hyperventilation and low carbon dioxide combining power may be a great factor in respirator habituation. Taking this into account has led us to take patients out of the respirator for short intervals of one to five minutes, starting as soon as respirator assistance is instituted. Time out of the respirator is gradually increased by 5 to 15 minute increments. Signs of distress and fatigue are carefully watched for, and a pulse rise of 20 points is considered valid reason for closing the tank. A measured increase in the ability to breathe is employed as an additional index of the patient's ability to withstand longer periods out of the respirator. Increasing the time out is carried on until finally the patient is out of the tank all day but returns to it at night. When a patient is able to withstand being out of the tank all day without undue fatigue or significant drop in vital capacity he is then ready to be taken completely out of the respi-

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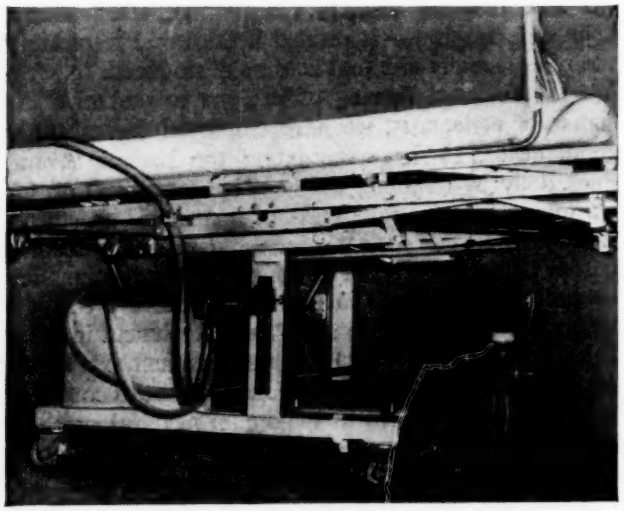
rator with the clear understanding that he will be returned for intervals of rest if his breathing capacity decreases. Such has occasionally been necessary.[7]

ROCKING BED

Intermittent daily use of the rocking bed has been of great value in decreasing respirator dependence once the acute phase of illness is past. It is wise to keep in mind that the amount of tidal exchange produced by rocking is below minimal requirements.[8] Patients with little or no breathing capacity, therefore, cannot be expected to ventilate adequately on a rocking bed unless additional aid to breathing is supplied. A 12-year-old boy who was quadriplegic for five months gave us the impetus to explore this field. He had a vital capacity of zero, and a serious renal and bladder infection had developed from prolonged urinary stasis due to his recumbent position in the respirator. It was not only felt that the rocking bed would facilitate the drainage of pulmonary secretions, but also that the postural change would beneficially effect



An original adaptation of the intermittent positive pressure respirator attachment to the rocking bed.

renal drainage. Within three minutes after being placed on the rocker the patient was in respiratory difficulty. The idea was then conceived that an intermittent positive pressure respirator attachment could be adapted to the rocking bed and thereby the efficiency of this artificial respiratory unit could be increased so that it would produce an adequate tidal exchange (see figure).

The adaptation was very simple. Directly under the bedsprings a section of ¾ in. pipe was bracketed to run longitudinally from the center of the bed to the foot. A piece of angle iron was then bracketed to the lower portion of the supporting framework for the bed in such fashion as to run below the pipe. With the bed horizontal the pipe and the angle iron ran parallel in the vertical plane. The positive pressure bellows was then placed vertically between these two supports and clamped in position under the foot of the bed. A lever-type snap clamp was attached to the outside of the bedspring frame near the head of the bed, to afford support for the long plastic tube that conducts air from the bellows to the tracheotomy. As the foot of the bed rocks down, producing inspiration, the bellows is compressed and forces air via the plastic tube down the tracheotomy. The amount of air delivered can be varied by moving the bellows from the point of least excursion near the fulcrum or rocking-axis of the bed to any desired point further out toward the foot, where excursion is greatest. A tidal exchange of 700 cc. can be produced in this fashion. We have employed this device in four tracheotomy patients with gratifying results. Patients with no respiratory ability have repeatedly been maintained in good condition for 12 hour periods. Such an apparatus has a limited but useful application.

TRACHEOTOMY

To facilitate respirator care our team has adopted the technique of early tracheotomy at a high level—preferably through the second tracheal ring—in those patients presenting the indications for tracheotomy. The high level is necessary to afford working room for tracheotomy care.[1] Local anesthesia has proved adequate for the procedure, during which assistance to respiration is maintained by manual compression of the breathing bag of a standard anesthesia machine. The decision to adopt so-called elective tracheotomy was reached after it became apparent that several patients treated by the conservative methods of postural drainage and suction[9] might have fared better with early tracheotomy.

We were influenced by other factors as well. First, it would seem that any patient possessed of a progressive disease that makes him incapable of swallowing his own secretions is most precariously situated.[10] Such a patient is apt to vomit or regurgitate, and a single episode of this sort could cause death. Second, the initial problem of upper respiratory obstruction, hypoventilation, and asphyxia, with resultant pulmonary edema and death, would appear more safely combated by the positive approach of tracheotomy.[11] Third, lower respiratory obstruction and atelectasis can best be prevented by tracheotomy in that group of patients who have or may progress to a breathing capacity of zero, even in the absence of bulbar involvement.[12] Use of an intermittent positive pressure respirator attachment with tracheotomy makes it possible to have the tank open long enough to perform the nursing necessities and still maintain breathing. Additionally, it is possible to clear the lower respiratory passages at any time without patient cooperation. This is vitally important. Tracheotomy is not without the many hazards of pneumothorax, bleeding, plugging, vocal cord injury, and infection[13]; however, the advantages gained by its judicious and timely use far outweigh the disadvantages.

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8. Bryce-Smith, R., and Davis, H. S.: Tidal Exchange in Respirators, Anesth. & Analg. **33**: 73-85 (March-April) 1954.
9. Brown, I. A., and Baker, A. B.: Treatment of Bulbar Poliomyelitis, Postgrad. Med. **9**: 151-155 (Feb.) 1951.
10. Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice, ed. 2, Baltimore, Williams & Wilkins Company, 1939, p. 679.
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12. Davis, H. S., and Bishop, H. F.: Airway Management in Patients with Poliomyelitis, J. A. M. A. **149**: 1175-1180 (July 26) 1952.
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LABORATORY STUDIES

Our experience has demonstrated the need for repeated blood transfusion, plus daily administration of plasma and concentrated serum albumin, particularly during the first 10 to 21 days of hospitalization, because anemia, decreased total serum protein, and reversal of the albumin-globulin ratio have shown a marked tendency to develop in our patients. The deviations from normal have paralleled the degree of illness and have led us to employ careful laboratory control of blood chemistry studies in the acute phase. The studies ordered are (1) albumin-globulin ratio, (2) total serum protein, (3) red blood cell count, (4) hemoglobin, (5) hematocrit, (6) carbon dioxide combining power, and (7) serum chloride. These studies are done daily in many of the critically ill and are progressively decreased as the patient's condition stabilizes.

If any single laboratory test could be chosen as being of paramount importance that choice would be a daily determination of the albumin-globulin ratio, since the reversal of this ratio and depletion of total serum protein occur so commonly in the patient with extensive paralysis. Add to the inflammatory edema of the central nervous system a superimposed nutritional or hypoproteinemic edema and grave difficulty can ensue. Nerve cells may be made physiologically inactive from the edema alone. If allowed to persist, this edema may destroy nerve cells never touched by the virus.

RESULTS

During three consecutive years of poliomyelitis team operation 51 patients were treated who required mechanical aid to respiration for an average of 114.3 days. The tank-type respirator was employed in 49 cases. An intermittent positive pressure respirator attachment was used on all respirators with tracheotomy patients. Two patients in the acute phase were satisfactorily treated by use of the rocking bed. One of these had a tracheotomy through which inspiration was further augmented by means of an intermittent positive pressure respirator attachment specially adapted to the rocking bed. There were 7 bulbar cases, 10 high spinal, and 35 bulbospinal cases. In this critical group 30 tracheotomies were done, or an incidence of 58.8%. There were 12 deaths, giving a mortality rate of 23.5%.

SUMMARY AND CONCLUSIONS

Using a poliomyelitis team, with the anesthesiologist responsible for the respirator unit, 51 patients requiring respiratory assistance were treated over a three year period with a mortality rate of 23.5%. Two patients below three months of age required the respirator; both recovered. The adaptation of an intermittent positive pressure respirator attachment to the rocking bed was used for four patients who required long-term respirator assistance.

It might well be said that patients with poliomyelitis most often die of inadequate and poorly timed treatment. Prohibitive mortality rates can be lowered by a planned approach on the basis of an organized "poliomyelitis team," whereon the anesthesiologist can play a vital part. Strict attention should be given to the principles of early patient transport to treatment facilities, early tracheotomy at the second tracheal ring, serial ventilation studies as a guide to early respirator care, and rigid laboratory control of blood chemistry studies.

505 Chestnut St. (Dr. O'Brien).

CLINICAL NOTES

EVALUATION OF AN ORTHOTOLIDINE TEST (FECATEST) FOR DETERMINATION OF OCCULT BLOOD

J. Alfred Rider, M.D., Ph.D., San Francisco
and
Francis J. Owens, M.D., Chicago

For practical purposes, normal persons on a meat-free diet do not pass occult blood in the feces. Consequently, the presence of occult blood with the absence of bleeding from the mouth, throat, respiratory tract, or anal ring indicates an ulcerating lesion in the gastrointestinal tract, very often peptic ulcer or carcinoma. Positive tests for occult blood in the feces may be the earliest indication of such lesions.

The four principal methods for the detection of occult blood in the feces have been reviewed by Hughes.[1] They are (1) microscopic demonstration of red blood cells, (2) microscopic demonstration of crystals of hemoglobin or derivatives, (3) spectroscopic tests demonstrating the presence of hemoglobin or derivatives, and (4) tests depending on the ability of hemoglobin or its derivatives to oxidize certain chromogenic substances (guaiac, guaiaconic acid, aloin, benzidine, malachite green, phenolphthalein, and orthotolidine). In this latter group greatest attention recently has been devoted to the guaiac, benzidine (including modifications), and orthotolidine tests.

In vitro studies have shown that the maximum dilution of blood detectable for the guaiac test ranges from 1:1,000 [2] to 1:30,000 [1]; for the benzidine tests, from 1:48,000 [1] to 1:5,000,000 [3]; for modified benzidine tests, 1:16,000 [1] to 1:100,000 [4]; and for the orthotolidine test, 1:20,000 [2] to 1:1,000,000.[5] The amount of ingested blood sufficient to cause a positive test for occult blood in the stool is small for all three methods. Peranio [6] in a review of the literature found that orthotolidine gave a positive test for 1 to 4.5 cc. of ingested blood; benzi-

From the Department of Medicine, University of Chicago. Dr. Rider is now at the University of California School of Medicine.

Dr. Joseph B. Kirsner, Professor of Medicine, University of Chicago, aided in the study and the preparation of the manuscript.

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5. Free, A. H.: Personal communication to the authors.
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dine was positive for 1 to 5 cc.; and guaiac was positive for 0.5 to 10 cc. Twenty cubic centimeters of ingested blood was required to cause a positive guaiac test with the patient on a meat-free diet, while only 2 to 3 cc. sufficed with an unrestricted diet. Cowie [7] found that as little as 20 gm. of uncooked beef in the diet caused a positive guaiac test. The sensitivity of the tests also can be increased by the use of laxatives. Other sources of error [8] are the presence of ferrous sulfate in the diet, producing a weakly positive guaiac test, and soya beans and urease tablets, which when cold cause positive reactions to both the guaiac and benzidine tests. False reactions caused by soya beans and urease tablets do not occur, however, if these materials are first boiled. A solution of 1:1,000 copper sulfate also may cause a false positive benzidine test.

The benzidine test is the simplest and most accurate procedure available for the chemical detection of blood in the feces. This test, however, requires a meat-free diet, an inconvenience increasing the problem of cooperation from the patient. (Hoerr found that 95% of 92 unselected hospital patients' stools were positive to the benzidine test). Modified, less sensitive benzidine tests have been devised and may be used in patients not on a meat-free diet. Hughes [1] describes a modified benzidine test on 350 stools of patients eating an ordinary hospital diet with meat; positive stools were obtained in only 3, and there was a reasonable explanation for each. These tests may be criticized as not sufficiently sensitive, and one may be lulled into unwarranted security with a false negative reaction.

Hoerr [2] examined the first stool of 140 unselected hospital patients with the guaiac test and found 39 positive. There appeared to be no adequate clinical explanation for eight of the positive reactions. Furthermore, among 101 patients with negative reactions there were 3 patients with ulcerative malignant lesions of the gastrointestinal tract. The author concludes that negative reactions do not rule out organic disease of the gastrointestinal tract. Also, the number of false positive results was rather high.

Barnett [9] found that one-half of 24 patients with cancer of the gastrointestinal tract failed to have a positive guaiac test, indicating that this procedure should not be considered a "screening" test for cancer. He also found 5.7% false positive reactions in testing 255 stools from 168 patients with no dietary restrictions. There were 3.5% false negative reactions in patients with lesions that could be expected to bleed at the time of the test. Mason and Belfus [10] found that only 25, or 22.5%, of 111 patients on a regular diet who had a positive guaiac reaction had a positive benzidine reaction when tested after they had been on a meat-free diet; 4 of 10 patients with gastrointestinal cancer had a negative guaiac test.

These observations indicate that there are too many false negative reactions with guaiac and that the unmodified benzidine test is too sensitive for use with an unrestricted diet. The modified benzidine tests may be better, but since their sensitivity is similar to guaiac they also may produce too many false negative reactions. The objective in the examination of the feces for occult blood is simplicity without loss of accuracy; its accomplishment would facilitate the cooperation of the patient and the more universal testing of stools for occult blood.

An orthotolidine test (Fecatest) package [11] has been developed, which is designed for use with an unrestricted diet and can, if desired, be used by the patient at home in testing for occult blood. This paper will describe the results of this test in 100 unselected gastrointestinal clinic patients with and without a meat-free diet and compare these results with those of the benzidine test obtained while the patient was on a meat-free diet.

MATERIALS AND METHODS

Benzidine Test.—A saturated solution of benzidine is prepared by adding a pinch of powdered benzidine to 4 cc. of glacial acetic acid. To this is added 8 cc. of hydrogen peroxide. Using a pipet, 1 or 2 cc. of the solution is allowed to flow over the inside surface of the stool sample. A positive reaction varies in intensity from a pale blue or blue-green that develops slowly over a few minutes, recorded as 1+ reaction, to a deep blue-black color appearing almost at once, recorded as a 4+ reaction; 2 and 3+ reactions are arbitrary divisions between these limits.

Orthotolidine Test.—There are two components for the orthotolidine test, one an effervescent mixing tablet and the second a porous paper packet containing the blood-detecting reagent. The effervescent mixing tablet contains sodium bicarbonate and citric acid, which interact when placed in water to produce carbon dioxide. The effervescence merely stirs the contents of the toilet bowl after a stool has been passed so that blood present in the stool is distributed throughout the water in the toilet bowl.

The porous packet contains a mixture of orthotolidine, strontium peroxide, tartaric acid, and calcium acetate. The mechanism by which the material reacts with blood is not completely understood, but it is believed to be as follows: Blood is adsorbed on the surface of the packet, where it acts as a pseudoenzyme and catalyzes the breakdown of hydrogen peroxide to water and active oxygen. The active oxygen oxidizes the orthotolidine to a blue-colored oxidation product, which is also adsorbed on the surface of the packet, giving it a blue color. The hydrogen peroxide originates from the interaction of the strontium peroxide, tartaric acid, and calcium acetate of the powder mixture.

The specific instructions to the patients are as follows: The test is done in a toilet bowl that contains a bowel movement. (If possible the urine is passed first and the toilet is flushed before the bowel movement. The toilet should not be flushed again until the test is completed.) The white wrapper is opened, and the tablet is dropped into toilet bowl. The tablet should bubble until dissolved (about two minutes). The silver wrapper is opened by cutting or carefully tearing along the sealed edge. The paper packet inside must not be damaged. The packet is removed. It is held by one corner and

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8. Hughes.[1] Hoerr and others.[2]

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11. The test was developed by the Ames Company, Inc., Elkhart, Ind.

dropped into the toilet bowl. The packet should be completely wet and floating. The color that forms on face of packet is noted. Any color seen at end of one minute, two minutes, and four minutes is recorded on the test record. (The face of the packet may remain grey or tan or become stained with blue.)

PROCEDURE AND RESULTS

Each of the 100 patients was instructed to eat a meat-free diet for six days. A stool was tested each day at home by the patient according to the above instructions. Portions of the tested stool from the last three days then were tested with benzidine in the laboratory.

The great majority (80%) of the patients were able to test all six samples; a few tested only four or five. Those who were unable to test at least four stools were not included in the series. Since several days are required to rid the gastrointestinal tract of previously ingested meat, it was possible to compare the orthotolidine test for the patient on an unrestricted diet (the first two or three stools) with that for a patient on a meat-free diet (the last three stools). In addition, this test could be compared to the benzidine test on a meat-free diet. Thus, the strictest comparison was possible. All patients had a complete gastrointestinal x-ray survey as well as a proctoscopic examination in order to confirm the diagnoses.

Of the 100 patients there were 49 with a negative orthotolidine and a negative benzidine test; none of these patients had organic gastrointestinal disease. There were 11 patients who had a positive orthotolidine test and a positive benzidine test; all of these had organic disease adequately explaining the positive results. There was one patient with a negative orthotolidine test but two trace positive benzidine tests for which there was no explanation.

There were 39 patients in whom there was no correlation between the orthotolidine test and benzidine test; all had positive orthotolidine tests. Nine positive tests were adequately explained on the basis of hemorrhoids, rectal polyps, or ulcerative colitis, since by virtue of the mechanics of the test benzidine did not give a positive reaction when there was blood only on the surface of the stool. There were 14 patients who gave a positive orthotolidine test during one or all of the first three days only. It is felt that these can be explained on the basis of diet. There were 11 patients who had from one to six trace reactions; these usually were described as very faint blue reactions or small (in some cases pinhead size) blue spots on the testing paper. These trace reactions were found not to be caused by organic disease. Whether they were caused by a reaction with urine, the water in the toilet bowl, or something in the diet is unknown. There were five additional patients who had from one to four positive orthotolidine tests (13 of the 30 tests in this group); these could not be explained by diet or by obvious rectal or other gastrointestinal pathological conditions.

SUMMARY AND CONCLUSIONS

For practical purposes the orthotolidine test can be used by the patient at home while he is on an unrestricted diet. However, if any positive reactions occur, it is essential to have the patient go on a meat-free diet and then repeat the tests. If the trace reactions to the orthotolodine test are excluded, there are 19% false positive reactions. If the stools from the patients on the unrestricted diet are excluded, this figure becomes 5%. Thus, in the present series 5% gave false positive reactions for the orthotolidine test, but there was only one false positive reaction for the benzidine test. There were no false negative reactions for either the orthotolidine or the benzidine test.

The orthotolidine test appears to be an excellent simple screening test for occult blood in the feces, providing the patients are on a meat-free diet and trace reactions are excluded. The incidence of false positive tests in the present series of 100 unselected gastrointestinal patients was 5% compared to 1% for the benzidine test. Lesions low in the colon and rectum causing blood to be present on the surface of the stool increase the incidence of positive orthotolidine tests compared with the benzidine procedure, in which only the inside of the stool is examined.

ACCIDENTAL POISONING BY VERATRUM JAPONICUM

A CLINICAL REPORT

Lieut. Dewey A. Nelson (MC), U. S. Army

On May 6, 1953, while serving as a battalion surgeon in Korea, I was contacted by a Korean Service Corps physician to observe and advise treatment to 14 men. During the preceding winter months the diet of the Korean Service Corps had been very deficient in leafy vegetables; on the first appearance of green plants, the cook of a 14-man labor party had selected those he thought were edible and boiled them into a green soup (a common practice among Korean rural dwellers). The soup was eaten at 12:00 noon. At 1:00 p. m. three of these men began to vomit profusely, and two hours later they complained of seeing circles, yellow sheets of flame, and yellow and green spots before the eyes. At 3:00 p. m. six more men became ill and reported to their medical officer. In addition to severe vomiting, prostration, weakness, and the visual disturbances described above, they complained of being unable to move the eye from a position of forward gaze. At this point the Korean physician sought aid. Because I was unable to leave my aid-station, I advised that 1,000 cc. of isotonic sodium chloride solution be administered intravenously to each man.

At 5:00 I first observed these patients. During the next five hours, five more patients reported to the medical tent complaining of the above symptoms, which had begun in the early afternoon. This brought the total number of cases to 14. At this time I did not know the cause of these bizarre clinical manifestations; it was as-

From the United States Army Hospital, 8079 Army Unit.
Lieut. Nelson is now with the 14th Medical Dispensary.
Capt. Edwin W. Smith, Dental Corps, U. S. Army, Lieut. Paul L. Redfearn, Medical Service Corps, 406th Medical General Laboratory, and Dr. M. Fujita, Assistant Professor of Pharmaceutics, University of Tokyo Medical School, assisted in the preparation of this report.

sumed that a plant or plants had poisoned the patients, but the nature of these plants was unknown. It seemed logical, however, to attempt to elevate the blood pressures and speed the pulses by subcutaneous and intravenous instillations of epinephrine. Due to particularly adverse field conditions, it was difficult to keep a clinical record on every patient, although it was possible to record some blood pressures and pulses. The efficacy of therapy was judged in most instances by clinical judgment of the pulse characteristics. A Tycos sphygmomanometer was used. Without exception, the patients appeared extremely ill and had paroxysms of vomiting that produced green matter and copious quantities of fluids. All pulses were below 40 per minute; the most severely ill patient had a pulse of 4 to 5 per minute. All had injected conjunctivas, distended abdomens, and difficulty in opening the eyes widely. No extraocular paralyses were noted. The average blood pressure was 80/60 mm. Hg, the highest blood pressure before treatment being 105/60 and the lowest, 50/0 mm. Hg. Summaries of the courses of three patients over a 15 hour period are presented in table 1. The lag in time between initial observation and aggressive

TABLE 1.—*Clinical Summaries of Three Cases of Veratrum Poisoning*

Time	Pulse	Blood Pressure, Mm. Hg	Temperature, Degrees F	Clinical Observations	Treatment
			Case 1		
5:00 p. m.	32, regular	.....	...	Vomiting, lethargy, eyes squinted, conjunctivas injected, weak pulse, muscular weakness	Infusion of 1 l. isotonic sodium chloride solution ending
5:45	20, regular	.....	...	Pulse weak	1 cc. 1:2,600 epinephrine subcutaneously
5:50	44, regular	50/0	97.5	Semiconscious, does not answer	1 cc. 1:2,600 epinephrine subcutaneously
5:53	44, regular	50/40	...	Generalized muscular tremors; no response to painful stimuli	
6:15	4-5	0/0	...	Forehead and extremities cold, skin dry, respirations slow	0.5 cc. 1:2,600 epinephrine intravenously
6:17	54, regular	100/50	...	Attempts to arise; deep, labored respirations, extreme apprehension	
6:22	.....	50/40	...		
6:30	.....	50/0	...	Unresponsive, extremities cold	4 cc. 1:1,000 epinephrine added to 1 l. isotonic sodium chloride solution and infusion begun
6:40	56, regular	115/60	...	Responds to questions	Infusion continued: 150-160 drops per min.
6:45	54, regular	130/80	...	Severe vomiting and retching	Infusion slowed
7:30	60, regular	60/40	...	Complains of weakness	Infusion speeded
7:50	.....	120/70	...	Retching	Drops per min. decreased
8:40	.....	100/65	...	Asleep, awakens easily	
9:30	66	115/70	...	Asleep	Infusion discontinued
10:10	.....	80/40	...	Restless	1 l. isotonic sodium chloride solution with 4 cc. 1:1,000 epinephrine begun intravenously
4:15 a. m.	65	.....	...	Pulse strong, asleep	Infusion discontinued
7:15	70	90/60	...	Awake, cheerful; voided 1,000 cc. light yellow urine: specific gravity 1.028, no sugar or albumin, centrifuged sediment shows 10-12 leukocytes, 3 erythrocytes per high-power field	
			Case 2		
5:10 p. m.	50, regular	.....	100	Severe vomiting, injected conjunctivas; complains of yellow light in front of his eyes; abdomen flaccid and distended, headache, pulse strong	1 l. isotonic sodium chloride solution just ending
6:50	38, regular	80/40	...	Very lethargic; moving patient causes vomiting and retching	0.25 cc. 1:1,000 epinephrine subcutaneously
7:20	40, regular	80/40	...	Restless	0.25 cc. 1:1,000 epinephrine subcutaneously
7:40	48, regular	90/60	...	Quiet *	
			Case 3		
5:15 p. m.	56, regular	90/50	...	Same symptoms as in cases 1 and 2	1 l. isotonic sodium chloride solution given earlier
7:45	40, regular	80/70	...	Extremities cold; responds sluggishly to questions	0.5 cc. 1:1,000 epinephrine subcutaneously
7:48	.....	200/100	...	Markedly apprehensive, complains of pounding in his ears; pulse fast and irregular	
8:05	.....	100/60	...	Resting quietly *	
10:15	70, regular	100/70	...	Asleep	

* The same therapy was repeated several times when the systolic blood pressure fell below 80 mm. Hg, but no accurate data were kept.

TABLE 2.—*Plants Contained in the Soup*

Generic Name	Common Name	Japanese Name	Korean Name	Toxic Property
Thalictrum bicaience	Meadow rue	Karomatsuso	Kongyechol Kori	None
Aralia elata	Devil's club	Taranoki	Turupnamu	None
Taraxacum albidum	Dandelion	Tanpopo	Pokongyong & Mumtore	None
Veratrum japonicum	False hellebore	Shuro-so	Paksai Ryoro	Toxic alkaloids
Aster scaber	Aster	Shira Yonogiku	Chui	None
Allium sp	Wild onion	Yama Negi	Manual	None
Adenophora verticellata	Unknown	Sobana	Tsugane Nengen	None
Carex siderostrica	Sedge	Taganeso	Unknown	None
Cimicifuga foetida	Snakeroot	Koraishoma	Probably known as Sunguma	None
Lathyrus davidii	Vetch	Endoso	Wharyong Namul	None

treatment was due to sending for medical supplies. The following morning all the patients had systolic blood pressures between 90 and 120 mm. Hg, and five of them returned to work. The cook returned to the area where the plants had been collected and picked a group that, he stated, were the very ones he had used the preceding day. I submitted these to the 406th Medical General Laboratory in Japan for identification. The only plant submitted that is suspected to contain toxic materials was Veratrum japonicum (table 2).

COMMENT

Veratrum nigrum L. var. japonicum (Baker) grows on the hills and mountainsides of Korea and Japan and, probably, in other Oriental countries with temperate climates. There is little clinical information concerning this variety, but the close parallel between the observed symptoms and symptoms of V. viride intoxication [1] suggests that V. japonicum produced these clinical manifestations. Muenscher [2] gives the habitat of the poisonous veratrum plants in the United States and lists symptoms of veratrum poisoning in domestic animals. It is interesting that these symptoms almost completely parallel those listed in this paper. V. viride is described as flourishing in the northeastern United States in low meadows and pastures, chiefly along streams; it also grows in the western states in the mountains. Another poisonous species, V. californicum Dur., is described by Muenscher [2] as occurring in wet meadows in the valleys between higher mountains.

The practice of picking "wild greens" in the springtime is not limited to Korea but is also done among some rural folk in the United States. With knowledge of the clinical manifestations of veratrum poisoning and the habitat of the veratrum species, it may be possible to diagnose similar cases of poisoning among rural populations in the United States and other countries.

SUMMARY

Fourteen cases of poisoning followed the ingestion of a number of plants, one of which was identified as Veratrum japonicum. It is believed that the observed symptoms were mostly due to V. japonicum alkaloids. With a knowledge of these clinical manifestations, perhaps cases of accidental poisoning by veratrum plants can be diagnosed.

APO 3, c/o Postmaster, San Francisco.

1. New and Nonofficial Remedies, report to the Council on Pharmacy and Chemistry, Alkavervir, J. A. M. A. **151**: 820-821 (March 7) 1953.

2. Muenscher, W. C. L.: Poisonous Plants of the United States, New York, the Macmillan Company, 1943.

The Work of the Potter.—Dr. Howard Rusk, New York, in a recent lecture before the American Academy of General Practice said, "Sick people ask their God, 'why must I suffer?' Possibly the answer is in the work of the potter. Fine ceramic pieces are not made by setting clay out in the sun. They come only from the white heat of the kiln. In the firing process some pieces are broken, but those that survive the heat are transformed from dull clay into objects of priceless beauty. And so it is with the sick, suffering, and crippled people. Those who, through medical skill, opportunity, work and courage, survive their illness or overcome their handicap, take their places back in the world with a depth of spirit which we can hardly measure."

MECHANICAL HIP EXTENSOR

Louis N. Rudin, M.D.
and
Daniel J. Cronin, Fort Howard, Md.

In order to make a paraplegic ambulant with the aid of crutches, stability must be obtained at the knee and hip joint. This is accomplished by use of long leg braces with knee locks, hip locks, and a pelvic band. When, in addition, there are varying degrees of paralysis of the back and abdominal muscles, a back brace is added. The assumption of a sitting position presents a problem, inasmuch as the hip locking device, either a ring drop lock or a thumb device, must be released. Since the patient's hands are not free because he has to hold on to his crutches, someone else must release these locks to enable the patient to sit down. In getting up from a chair the patient supports his weight on his hands and arms, advances his trunk forward, and throws his abdomen anteriorly to lock the brace hip joint.

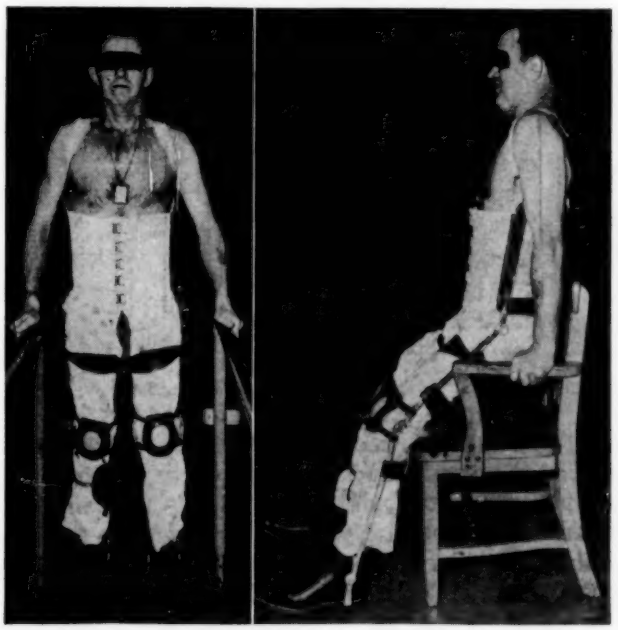

Fig. 1.—Paraplegic patient using the mechanical hip extensor as an aid in standing and sitting down.

In order to make a paraplegic independent of outside help in sitting down and make it easier for him to get up from a chair, a mechanical hip extensor has been devised (fig. 1). It consists of a coiled spring attached by posts to the pelvic band or back brace and side bar of the long leg brace (fig. 2). The tensed spring produces a constant extending force on the hip joint in a posterior direction. It prevents accidental sudden flexion at the hip (jackknifing) and falling when he is in the upright position.

Chief, Physical Medicine Rehabilitation Service (Dr. Rudin) and Supervisor, Orthopedic Brace Shop (Mr. Cronin), Veterans Administration Hospital.

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Mr. William C. Beck Jr., medical photographer, and Mrs. Jeanette M. Whitehorn, medical artist, Veterans Administration Hospital, prepared the illustrations.

When the patient sits down, the weight of his body overcomes the tension of the spring and brings it to about 90 degrees. During this movement the U bar of the Swiss knee lock hits the edge of the chair and opens the brace knee joint. Thus the patient is able to sit down and get up from a chair without anyone else or the patient handling manually the hip or knee locks. In getting up, the tensed spring is of material aid. Only moderate pressure

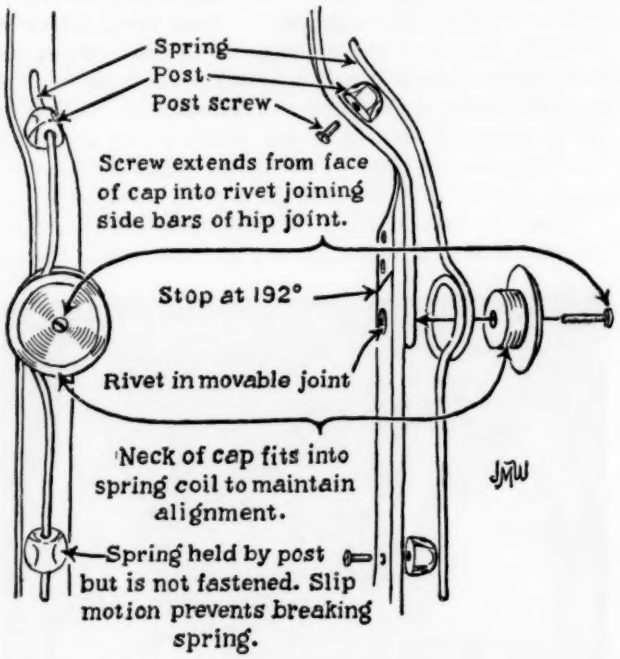

Spring
Post
Post screw
Screw extends from face of cap into rivet joining side bars of hip joint.
Stop at 192°
Rivet in movable joint
Neck of cap fits into spring coil to maintain alignment.
JMW
Spring held by post but is not fastened. Slip motion prevents breaking spring.

Fig. 2.—Drawing showing the parts of the mechanical hip extensor.

on the arms of the chair with the hands will bring the patient to an upright posture. The spring or springs give the patient a certain amount of flexibility at the hip joint in the standing posture, which is desirable. The mechanical hip extensor has been found to work satisfactorily either with a back brace or a pelvic band attached to long leg braces.

Jaundice Due to Testosterone.—There is increasing evidence that a distinct type of hepatic damage is produced in some patients by administration of methyl testosterone. . . . As we become more aware of jaundice as a possible effect of methyl testosterone, we may expect this condition to be recognized more frequently. The mechanism of the action of methyl testosterone in producing the liver damage remains obscure. When Kinsell called attention to methyl testosterone jaundice in 1948 he pointed out that methyl testosterone caused marked creatine formation and excretion, and that this may occur at the expense of choline, with a decrease in the production of phospholipid and the resultant fatty infiltration of the liver. . . . [He] reported that there was no evidence of fatty infiltration in specimens obtained by liver biopsy. He therefore concluded that there was no substantiation of the belief that the loss of creatinine resulted in loss of labile methyl groups that are considered to be instrumental in minimizing liver damage and fatty infiltration. . . . Methyl testosterone ingestion results in increased urine output of 17-ketosteroids, and offers the possibility that an intermediate metabolic product of methyl testosterone, rather than methyl testosterone itself, may be the icterogenic factor as well as the cause of creatinuria.—P. J. Almaden, M.D., and S. W. Ross, M.D., Jaundice Due to Methyl Testosterone Therapy, *Annals of Internal Medicine*, January, 1954.

SPECIAL ARTICLE

The following material was prepared by the Committee on Civil Defense of the American Psychiatric Association, at the request of the Federal Civil Defense Administration, as an aid to physicians for use in dealing with lay groups charged with civil defense responsibilities. It is being reproduced with the permission of the American Psychiatric Association because of the valuable information that it contains concerning the emotional needs of disaster victims and how those needs can be met. This material has been reviewed by members of the Council on National Defense and the Committee on Mental Health of this Association, and they have felt that even though much of the material presented is familiar to physicians that the compact presentation of this information as given here can be of considerable value to physicians who are or will be dealing with lay members of their local disaster relief organizations. A second manual, which will concern itself with the more professional aspects of the problems involved, is now in preparation. Additional copies of the prepared pamphlet from which this material is taken can be obtained from the American Psychiatric Association, 1785 Massachusetts Ave. N. W., Washington 6, D. C.

C. JOSEPH STETLER, *Secretary*
Council on National Defense
R. J. PLUNKETT, M.D., *Secretary*
Committee on Mental Health

PSYCHOLOGICAL FIRST AID IN COMMUNITY DISASTERS

PREPARED BY THE AMERICAN PSYCHIATRIC ASSOCIATION COMMITTEE ON CIVIL DEFENSE

Calvin S. Drayer, M.D., Philadelphia
Dale C. Cameron, M.D., Bethesda, Md.
Walter D. Woodward, M.D., New York
and
Albert J. Glass, M.D., San Francisco

WHY PSYCHOLOGICAL FIRST AID

First aid is just what its name describes. It is the first aid received by a person in trouble. Such aid should be concerned only with the immediate situation. First aid goals should be either to return moderately disabled persons to reasonably good function in a short time or to make the more serious casualties as comfortable as possible until more complete care can be arranged for them.

Most of you do not have much occasion to work with people who are grossly upset emotionally. In all disasters, whether they result from the forces of nature or from enemy attack, the people involved are subjected to stresses of a severity and quality not generally encountered. Their reactions to these experiences may be quite different from their usual behavior. It is vital for all disaster workers to have some familiarity with common patterns of reaction to unusual emotional stress and strain. These workers must also know the fundamental principles of coping most effectively with disturbed people.

Although this publication has been stimulated by the current needs for civil defense against possible enemy

action, the principles described in it are widely applicable to any community disaster. These principles are essential for those who are to help the victims of floods, fires, tornadoes and other natural catastrophes.

FIVE TYPES OF REACTIONS TO DISASTER

Knowledge of a few basic surgical principles enables many nonmedical people to give first aid for physical injuries. Similarly, awareness of certain psychological facts will greatly simplify your care of terrified, depressed, disaster victims. Whatever your special disaster assignment may be, you will inevitably be working with people under great stress. The following are the general types of reactions to be expected in varying proportions according to the nature and magnitude of the disaster:

Normal Reactions to Disaster.—Some people are able to remain remarkably calm, at least for a time, even under the most extraordinary circumstances. The great majority of us, however, tend to show obvious signs of disturbance. We may perspire profusely, we may tremble a bit, or we may even feel weak and nauseated for a little while. Clear thinking may be difficult for a time. Fortunately, many of us can regain our composure fairly well rather soon after the first impact of a devastating experience. It would be misleading to classify all these natural and largely transient reactions to stress as abnormal. There are occasions, however, when even very stable people are so overwhelmed by an event, that they are unable to recover a semblance of emotional balance for quite a while and may require help from someone else.

Individual Panic (Blind Flight).—Although this reaction to danger is far less frequent than you may expect, its notorious contagiousness gives it an importance far out of proportion to its initial frequency. A very small number of individuals in true panic can easily precipitate headlong mass flight of a crowd.

The outstanding characteristic of panic is its blindness. All judgment seems to disappear and to be supplanted by an unreasoning attempt to flee. On a sinking ship, overcrowded lifeboats have been stormed by terrified passengers who seemed totally unaware of other less-filled boats nearby. In theater fires the risk of being crushed to death has not deterred some in the audience from stampeding a single exit, even though other exits were obviously available.

In other instances, individual panic may be manifested by pointless physical activity, such as uncontrolled weeping or wild running about. Situations most conducive to this type of behavior (often called "hysterics") are those where known escapes actually are, or are believed to be, threatened by progression of the danger. Sheer horror may also precipitate a very similar reaction. The sight of close friends or family who have suffered gruesome mutilation may bring on wildly disorganized behavior in certain persons, especially if they themselves have suffered little or no physical injury.

One must remember, however, that simply moving away from a source of real danger as rapidly as possible, and in an orderly fashion, is not evidence of panic. If no useful purpose is to be served by remaining in a perilous situation, constructive efforts to escape from it are only manifestations of good sense.

Depressed Reactions (Slowed Down, Numbed).—In disasters many people will act for a time as though they were numbed. They may stand or sit in the midst of utter chaos as though they were completely alone in the world. Their gaze will be vacant. When spoken to, they may not reply at all, or simply shrug their shoulders and utter a word or two. Unlike the person in panic who seeks physical escape at any cost, they appear to be completely unaware of the situation and devoid of emotional reaction to it. They are unable to help themselves without guidance. After the recent earthquakes in Greece, many survivors were found doing nothing, even though tools for reconstruction were at hand in their damaged communities. Such disaster victims are obviously unable to take responsibility for themselves or for jobs requiring any initiative. Fortunately, a great many of the less severely disturbed persons in this group can be salvaged rather quickly and with fair success by methods described later in this manual.

Overly-Active Responses.—In contrast to the disaster victims just described, others will explode into a flurry of activity which at first glance may seem purposeful, but will soon be recognized as largely useless. They will talk rapidly, joke inappropriately, and make endless suggestions and demands of little real value. They will jump about from job to job and seem unable to resist the slightest distraction. They appear to flee into an unreal confidence in their abilities, which causes them to be relatively intolerant of any ideas but their own. Consequently, even one such person can become a disturbing nucleus of opposition to sounder procedures than he himself can actually propose.

Bodily Reactions.—Some bodily reactions have already been mentioned as the normal temporary responses of many of us to unusual stresses. Although these "normal symptoms" are troublesome while they last, they generally do not interfere seriously with a person's ability to carry on constructive activity in a difficult situation. Certain of these reactions, especially weakness, trembling, and crying may not appear until after a person has effectually met and surmounted the immediate danger.

More serious and sustained bodily reactions can be truly disabling, however, and it is important to recognize their emotional significance. Severe nausea and vomiting are particularly common. Since these symptoms may also result from serious exposure to radiation or to certain biological and chemical warfare agents, it should be remembered that many individuals *not* so exposed may suffer from these symptoms for purely emotional reasons.

Another type of bodily disability may be encountered which is basically different from the disorders just mentioned. This is technically called "conversion hysteria" (not to be confused with the condition of wild excitement usually designated by the popular term "hysterics"). In conversion hysteria, the person *unconsciously* converts his great anxiety into a strong belief that some part of his body has ceased to function. For all practical pur-

poses he may be unable to see, or to hear, or to speak. All feeling or power may disappear from one or more limbs. The development of such conversion symptoms reduces the overwhelming anxiety previously felt. It should be emphasized that such casualties are not "faking" (malingering). They are completely unaware that no physical basis for their symptoms exists, and they are just as disabled as though they had a physical injury.

The disaster reactions described above have been separated into five categories largely for the sake of clarity and because, to some degree, individuals in each group obviously must be treated differently when encountered. Actually one disaster victim may show some features of more than one category simultaneously or at successive stages of his reaction. For example, the initially numbed, depressed victim may later swing spontaneously into excessive activity and great distractibility. These variations need not be too troublesome in psychological first aid, however, since your chief job will be to deal with the casualty as you find him and for only a relatively short period of time thereafter.

FOUR BASIC PRINCIPLES

To be of maximum service to an emotionally disturbed person, certain general principles must be reasonably understood and believed. A brief discussion of them follows:

Accept Every Person's Right to Have His Own Feelings.—Do not blame or ridicule a person for feeling as he does. Your job is to help him cope with his feelings—not to tell him how he *should* feel. If you stop to think of your own feelings, you will realize how impossible it is for human beings to make a conscious choice of their deeper feelings. Our freedom of conscious choice—and hence our responsibility—depends upon what we *do* about our feelings to relieve the tensions they create within us. After we have taken appropriate action, the initial feelings may change, sometimes quickly, sometimes gradually. Nothing is gained by trying to deny that the initial feelings existed just because they were at variance with what we now think a self-respecting human being *should* feel.

Keep in mind that however similar another person's background may be to your own, each person has had certain unique experiences which can strongely affect his feelings in relation to subsequent events in his life. Thus disaster will have many meanings to the many victims. Above and beyond the horror and fear which all may properly feel, the person will react in terms of how this experience fits in with his past ideas about himself and his aspirations for the future, as well as the world in which he has lived and expects to live.

All of us have failed in some things we have tried to do and have succeeded in others. Being human, we have not always really understood just why we have failed or succeeded. Hence we have looked about us and within ourselves for understandable "reasons" for these failures and successes. It is a rare person who can accurately identify the causes and motivations of actions in which he is involved or his own motivations concerning them. The average person tends to give more blame or credit than he should to himself, to others, or to chance. It is the rule rather than the exception that people are either more proud or more ashamed of courses of conduct than the actual facts entitle them to be. When disaster strikes, many will be inclined to suspect, at least, that their past mistakes account in some way for their being among the victims.

A poignant example of this trait has been reported from Nagasaki, where the second atomic bomb attack was so devastating. A little boy had been caught beneath a fallen beam and could not free himself because a spike pressed on his chest. He later recalled the first thoughts he had about the *meaning* of the experience to him. He was *sure* the spike on his chest was a direct punishment, designed specially for him, because he had collected butterflies and pinned them to a board!

In giving psychological first aid, you will seldom be exactly sure *why* some persons become more disturbed than others about them. Years of research and analysis may sometimes fail to explain fully *why* a person feels as he does. Fortunately, it is infinitely easier to determine *how* he feels at a given moment. His expression, his posture, his whole manner can tell you this, even though he says nothing with words. Letting a casualty know you want to understand *how* he feels can be the first step toward helping him. This may be done with a few words or even a simple gesture. Do not overwhelm him with pity. This will only make him feel more inadequate and will confirm his worst fears about himself. Just do enough to establish such contact as you can with him. If he senses even vaguely that you are trying to see this disaster through his eyes, your chances of helping him find constructive outlets for his distressed feelings will be greatly increased. It is the collapse of *his* world that distresses him. He will not trust you very far if you deny his world by telling him he has no need to feel the way he *knows* he feels.

Accept a Casualty's Limitations as Real.—When a man's thigh is shattered, no one (including the patient) expects him to walk for a time. When a man's ability to cope with his feelings is shattered, many (often including the patient) are inclined to expect him to function normally again almost immediately. "It's all in your head." "Snap out of it." "Pull yourself together." Such goading, scolding types of "reassurance" have no place in psychological first aid. The casualties you meet will not *want* to feel as they do. They will want to be as effective as possible; but their feelings of incompetence or overconfidence are real and often disabling. It will be your job to help them regain as much effectiveness as they can as rapidly as they can. This is best done by accepting the handicaps they present, and by helping them rediscover quickly a few of their assets which they can use at once.

In doing this, you must at all costs guard against a very common human tendency in yourself. Especially when you are as fatigued and as frustrated as you may well be in the midst of a disaster, you can easily begin to resent intangible disabilities in those around you. Physical wounds you can see. Anxiety in others is harder to rec-

ognize. After all, you will feel, you have managed to get yourself passably well reorganized. Why cannot others? Why do they need your help to do for themselves what you believe you have managed independently for yourself? Again you need to recall that the same experience can have vastly different meanings for different people. Even though you may be able to restore some semblance of order in your private world fairly rapidly after a disaster, others may well find the job far more complicated. They deserve your understanding help—not your resentment—in response to their very real needs.

Size up a Casualty's Potentialities as Accurately and as Quickly as Possible.—While you are making allowance for a disturbed person's limitations, you should be on the lookout for skills and other assets you can revive and utilize. A very upset person can easily cause you to forget that he *could* be of real assistance to you as well as to himself, if you can help him get started on the way toward reorganizing his world. Inquire gently into what happened to him. Let him reply in his own way. You cannot let him ramble on endlessly; but even a couple of minutes in which he can talk freely of his own experiences will relieve remarkably some of his feelings of despair and helplessness. Find out if a casualty is particularly concerned about family or friends. Give him as honest an estimate as you can of where, when, and how he may reasonably expect to make contact with those who are dear to him. If he is too depressed to talk easily, some statement of what *may* have happened to him (based on your general knowledge of the disaster) can increase his confidence in you to the point where he can talk a bit.

Brief questions about his normal occupation may give you some clues to his interests and basic abilities. For example, a man with manual skills can be asked to help you straighten up the immediate area where you are trying to do your job. A man with some clerical background may be of great assistance in simple jobs involving messages or records. For many persons who have been overwhelmed emotionally, work which can be done almost automatically and by rote can be the first step back toward normal effectiveness.

It is surprising how close you can get to a person under disaster conditions in a short time. Be warmly interested but not enveloping or maudlin. Treat him as a potentially valued member of your disaster team. Show your respect for him as sincerely as you can. Help him to find a way to utilize his skills as quickly as possible.

Accept Your Own Limitations in a Relief Role.— Do not try to be all things to all people. There will be much you would like to do in a disaster beyond your strength and skill. You cannot be all things to all victims. Consequently, you must establish a set of priorities in your thinking about what you will allow yourself to undertake. Your first responsibility will be to whatever emergency job you have previously been assigned. In theory, this may well be a full time job, but in practice, you will be under pressure to do some of the jobs assigned to others who may themselves be casualties. Here you must know yourself quite well to judge accurately what would be worth your trying and what would be a total waste of time. You will naturally extend yourself to the limits of your capacities. Be as sure as possible that you do not push yourself beyond those limits, lest you become as ill as those who need your help.

A reasonably candid appraisal of yourself is a very important part of your preparation for psychological first aid. A physically injured first-aid worker may continue to do a good job of dressing the wounds of other casualties. But if you are emotionally "wounded" yourself, you will be greatly handicapped in providing help for others with emotional difficulties. In other words, know your own weaknesses well enough to handle them quickly in time of crisis.

It should be clearly understood that in a community-wide disaster you yourself could be as subject to personal anxieties as less formally trained workers. Your training should prepare you to handle your own emotional problems first—and promptly. For example, your natural concern about the plight of your own family and friends should be anticipated so consciously and fully that you will not find yourself suddenly and unexpectedly bogged down by these concerns when disaster strikes. If you have done your best in advance to provide protection for your family, your feeling of personal responsibility for them will be less. (See "HOME PROTECTION EXERCISES —A Family Action Program," published by the Federal Civil Defense Administration.) During training, your annoyances at imperfections in the disaster plans should be worked through fully enough so that in time of crises you do not find yourself overwhelmed by a flood of resentment at "the authorities" because they have not given you everything you feel you should have had to help relieve suffering.

You should examine carefully certain trends in your own "normal" behavior. Are you impulsive? Are you easily angered? Are you a bit more sensitive to criticism than the average person? Are you inclined to store up resentments instead of expressing them wisely and effectively? Do you tend to judge people according to their race, religion, or other group affiliation instead of as people? Are you prone to make promises beyond your actual strength and capacities because you cannot bear to say "No"? None of these characteristics may have caused you any serious grief thus far. In the midst of a disaster, any one of them could trip you up badly if you are so unaware of their presence that you have never developed any ways of modifying them.

The training you receive as a disaster worker will in itself protect you somewhat in time of stress. Experience in real disasters, such as fires, floods, and tornadoes, will make the value of your training much more significant to you. Knowing how to do an important job well is a widely recognized antidote for many types of anxiety.

Nevertheless, regardless of how thoroughly you are trained, it would be unwise to assume that you will be completely immune to all personal disturbance if the community you know should be torn apart suddenly by some tragedy such as an atomic explosion. Hence your first job in psychological first aid is to look to your own defenses. If you understand yourself reasonably well,

you may justifiably hope to endure and control your own anxieties in the midst of a community-wide disaster.

Summary.—To sum up, the basic principles of psychological first aid concern ways of establishing effective human contacts with disturbed, overwhelmed persons who have lost touch to some degree with the world as it is. Once such contacts are made, it becomes reasonably easy to help many of these people to take an active, contributory part in the simpler phases of emergency activities. Devastating as a catastrophe may be, the very needs it creates can be an asset to psychological first aid. Unlike ordinary life, a disaster engenders more urgent jobs than there are people to do them. Opportunities to regain self-respect and self-confidence are correspondingly greater. Psychological first aid can help many emotionally disturbed victims to take advantage of these opportunities and thereby get back into their stride.

SOME PRACTICAL SUGGESTIONS

Your successful application of these general principles requires practice. Just as you need active disaster experience to learn more about yourself when you are under stress, so such experience is the only means by which you can really learn the value of the principles that have been discussed. Nevertheless, some examples of how these ideas may be applied to the chief categories of reactions previously described should be mentioned.

Normal Reactions to Disaster.—Obviously little or no help from you is required by the persons who remain calm and effective in the face of emergency, even though they may experience rather transient symptoms, such as nausea, sweating, or other normal reactions to stress. A word of sympathy and encouragement in passing can be well worth while, but it will be important (as well as necessary!) for you to assume immediately after the impact of a disaster that *most* of the surviving casualties can regain at least some control of their emotions if given a little time to do so. The course of the reactions will soon make it clear which persons are becoming actually disabled by their disturbed feelings and which are having reasonable success in overcoming their initial internal turmoil. Subsequent events may alter the situation in particular people, but a good general rule to follow is that psychological first aid is necessary only for those persons who are manifestly losing control of themselves or are making no perceptible progress toward resumption of effective behavior.

Individual Panic (Blind Flight).—These cases will respond least to the application of the general principles that have been presented. Physically, these individuals will be so explosive in their behavior that you will have difficulty even in getting their attention. They will be disturbing to all about them, and they may very easily provide the nucleus for development of a general panic. Consequently, their early segregation and control is urgent. How you can attain these ends will depend largely on the facilities available to you. Gentle firmness should be tried first, but if this fails, get two or three others to help you take these casualties to the nearest medical aid station you can find in operation. Should you attempt this forcefully by yourself, the casualty will almost certainly break away from you and become even more disturbed.

If you find yourself isolated for any great length of time from all medical facilities, it would be wise to ask two or three fellow workers to remain with the person in panic until help arrives. In such an arrangement, physical restraint may have to be used for a time; this restraint should be firm but not brutal or punitive. The widespread belief that a casualty in panic can be jolted out of his confusion by slapping him in the face, by dousing him with cold water, or by other forms of abuse is unsound. Such tactics may interrupt the disorganized behavior, at least briefly. But in the midst of disaster tensions, striking a disturbed casualty will not only fail as a rule to control his panic but will greatly increase the anxiety of those around you who may feel on the verge of similar panic themselves.

With regard to your being justified in restricting two or three people to the temporary care of a single casualty, it should be emphasized that panic spreads easily. The prompt control of people who are becoming panicky will go far to offset the great dangers inherent in the development of such behavior in large numbers of people.

Depressed Reactions (Slowed Down, Numbed).— It is with these persons that you may hope to get the best results by the methods which have been suggested in this manual. A relatively short time spent with each of them, or even with a number of them gathered in a small group, will bring out desirable responses in many. As they sense your constructive interest, their private worlds no longer seen quite so desolate to them, and your individualized suggestions for simple, routine jobs they might undertake will have a chance of making a useful impression. Some will naturally respond more quickly than others, and your own limitations in time and energy will obviously prevent your working very long consecutively with any one of them. Nevertheless, if you can help even a few of them to begin functioning again, others will tend to rouse themselves and will be more amenable to suggestions when you can spare a few moments for *them*. A casualty may at first show no sign of response to you. Yet he will be forming at least vague opinions of you as a person, and his later reactions to you will reflect the quality of his initial impressions.

Elderly people and children who are victims of a disaster present many special problems. This is not the place to discuss the long-term phases of these problems. From the standpoint of psychological first aid, however, the methods just described will be reasonably effective with both the aged and the very young casualties. Older persons will tend to become more confused in a disaster than the young or middle aged, and hence cannot be expected to respond to understanding care as quickly or as well the younger group. The reaction of children, on the other hand, will often reflect the attitudes of the adults around them, and calm, realistic assurances will go far toward helping them recover some equanimity. It is obvious that both the aged and the children should be given as much quiet supervision as possible during the chaotic period immediately following a disaster.

Overly-Active Responses.—Here again, as with the panicky persons, you may at first have trouble just getting the attention of the overly-active group. Unlike the pan-

icky persons, however, they will establish a flighty sort of contact with you which will give you some opportunity to indicate your interest in helping them become useful. Under proper supervision, which will compensate somewhat for their great distractibility, these persons can gradually attain a reasonable degree of composure. Their need for physical activity will be considerable, so that jobs such as moving rubble or rescuing casualties would have value for them.

Since these persons feel much confidence in themselves, they may cause serious trouble. They will be inclined to criticize what they regard as the stupidity of authority in general, and may well be outspoken in their resentment of some person or group whom they blame for the disaster. Like panic, such "scape-goating" attitudes are extremely contagious in disturbed situations. If they are allowed to spread, they can lead to a very serious crisis.

Hence every effort should be made to dissuade them from destructive expressions for the moment and to encourage them to devote their excessive energies to cleaning up the mess at hand. There is no need to argue the rightness or wrongness of their views; there may even be some actual basis for their resentment. It is far more important to make clear to them, as diplomatically as you can, that once a disaster has occurred the urgent need is for such repair of the damage as may be possible. Responsibility for the disaster can be considered later.

Bodily Reactions.—You should not find it difficult to talk calmly with this type of casualty. It will probably be impossible for you to relieve them of their bodily disturbances immediately, whether they be of the type represented by nausea, vomiting, or weakness, or of the type represented by hysterical blindness, deafness, or paralysis. It is important, however, to make them feel your interest in them as individuals. You may then be able to find a number of small jobs which they can do in spite of their symptoms. By thus by-passing their disability to some degree, you may help a number of them to regain their composure gradually, while waiting for medical help.

SEDATIVES—A LAST RESORT

Do *not* administer sedatives to psychological casualties except as a *last resort*, and even then consult a physician, if possible, before you do so. The psychological casualty does not think clearly. However calming a sedative may be, it will inevitably add to his confusion and make him even more inaccessible to the methods of treatment which have been described.

You may wonder whether in some instances, such as a wildly panicky reaction, it would not be simpler and better to put the casualty to sleep with a sedative until medical help arrives. Unfortunately, so seriously disorganized a person usually requires very large and dangerous doses of sedatives to be put to sleep. It is not uncommon to see a paradoxical response in which a disturbed person becomes even more excited when given large doses of a drug which usually has a sedative effect. Consequently it is far better to rely on the approaches already discussed in attempting to help the casualty with emotional disabilities. Should you be forced to use sedation, make doubly sure that the casualty is clearly tagged with a record of any medicine you have given him and of the time it was given. Otherwise, if he is still disturbed, you or someone else may later give him more sedation that could be fatal in combination with the first dose.

IF YOUR BEST EFFORTS FAIL

The problem remains of dealing with those disturbed casualties who continue unresponsive to your best efforts. It has already been pointed out that the actually panicky casualties may need to be brought under trained medical care as promptly as possible. The depressed and those with severe bodily reactions who you cannot reach are less urgent cases. They may be handled in groups, temporarily, in first-aid stations, mass care centers, or in improvised hospitals. They should also be brought under medical care as soon as possible. While they are waiting, their physical needs (warmth, food, protection from further danger) should be met. Those in the overly active (not panicky) group, who will pay no attention to you, may have to be tolerated until their dashing about seriously disrupts the work of others. It may then be possible to persuade them to join those awaiting medical attention on the grounds that they are overworking themselves and seem to be in danger of collapsing. If this face saving approach fails, more drastic measures, similar to those recommended for control of panicky people, may have to be used.

MASS PANIC

Chief consideration has been given in this manual to the recognition and care of emotional problems in *individual* disaster victims. The emotional disturbances of large crowds may also become a serious burden in any disaster. Although the forces involved in these group reactions are related to the forces in the disturbed individuals, the handling of them requires controls and techniques of leadership beyond the measures for dealing with individuals discussed here.

Nevertheless, as you learn and apply the principles of sound psychological first aid, you will contribute tremendously to the prevention of crowd activity which, should it get under way, may easily become impossible to control by the most effective leaders. Your success in restricting the influence of one panicky person may very well be the chief reason why his less disturbed associates turn to constructive activity instead of rushing off blindly to their own destruction. Deft control of a single overly-active, resentful person who seems bent on punishing the scapegoats he holds responsible for a disaster, can do more to avoid the horrible results of riots than all the efforts of troops or police who might have to quell a mob determined to vent their anger on some official or on some minority group.

Community leaders, as well as military and police personnel, have quite enough to do during any disaster without having to exhaust themselves in efforts to control disorganized mob activity. With the proper use of psychological first aid, you can do much to spare them this additional burden.

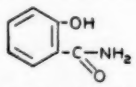
COUNCIL ON PHARMACY AND CHEMISTRY

NEW AND NONOFFICIAL REMEDIES

The following additional articles have been accepted as conforming to the rules of the Council on Pharmacy and Chemistry of the American Medical Association for inclusion in New and Nonofficial Remedies. A copy of the rules on which the Council bases its action will be sent on application.

R. T. STORMONT, M.D., *Secretary.*

Salicylamide.—$C_7H_7NO_2$.—M.W. 137.13.—The structural formula of salicylamide may be represented as follows:



Actions and Uses.—Salicylamide, the amide of salicylic acid, shares the actions and uses of acetylsalicylic acid (aspirin). Its analgesic potency is no greater and may be somewhat less than that of aspirin. Its antipyretic and anti-inflammatory or anti-arthritic properties are not superior to those of aspirin. The over-all incidence of gastric intolerance to salicylamide is about the same as, or a little less than, that to aspirin; however, patients allergic to aspirin have been reported not to be sensitive to salicylamide. It can be used safely in place of salicylates whenever such medication is indicated.

Salicylamide is readily absorbed from the gastrointestinal tract, but does not produce high salicylate levels in the serum. It is diffused widely throughout the tissues, excreted chiefly by the kidneys, and apparently is not destroyed appreciably in the body. Since the toxicity of salicylamide compares closely with other salicylates, it should be employed with the same general precautions. The possibility of the development of sensitivity to salicylamide after its repeated use, particularly in patients already allergic to other salicylic acid compounds, should be kept in mind.

Dosage.—Salicylamide is administered orally, preferably after meals and with fluids to minimize gastric irritation. The dosage should not be less than that for aspirin. As a simple analgesic or antipyretic, single doses of 0.3 to 1 gm. three times daily may be adequate; as an antirheumatic agent, doses of 2 to 4 gm. three times daily (or 1 to 2 gm. every four hours) may be prescribed, according to gastric tolerance, over periods of three to six days. For children, correspondingly smaller doses should be employed.

Tests and Standards.—

Physical Properties: Salicylamide is a white, almost odorless, crystalline solid, m.p. 139-142°. It is freely soluble in alkalis. The approximate amounts that dissolve at 25° in the following solvents to form 100 ml. of solution are 7 gm. in alcohol, 1 gm. in chloroform, 3 gm. in ether, 5 gm. in propylene glycol, and 0.2 gm. in water. Salicylamide is fairly stable to heat, moisture, and light.

Identity Tests: Dissolve about 0.1 gm. of salicylamide in about 5 ml. of alcohol, and add a few drops of ferric chloride T.S.: a violet color develops *(presence of a phenol).*

Dissolve about 0.2 gm. of salicylamide in 5 ml. of alcohol, and add 15 ml. of water. To this solution, add slowly and, with shaking, bromide T.S. until a yellow color persists. Allow the mixture to stand at room temperature with occasional shaking for 30 minutes, collect the precipitate, wash with water, recrystallize from aqueous alcohol, and dry in a vacuum over phosphorus pentoxide for 4 hours: the compound melts at 183-185°.

A 0.001% solution, prepared as directed in the spectrophotometric assay for salicylamide, exhibits ultraviolet absorption maxima at about 236 and 302 mμ [specific absorbancy, E(1%,1 cm.), about 302 and 548] and minima at about 224 and 263 mμ. The ratio of the absorbancies at 236 and 302 mμ is 1.77-1.87.

Purity Tests: Dry about 1 gm. of salicylamide, accurately weighed, at 105° for 4 hours: the loss in weight does not exceed 0.5%.

Char about 1 gm. of salicylamide, accurately weighed, cool the residue, add 1 ml. of sulfuric acid, heat cautiously until evolution of sulfur trioxide ceases, ignite, cool, and weigh: the residue does not exceed 0.05%. Save the residue for determination of heavy metals.

Dissolve the residue from the sulfated ash determination in 23 ml. of water, add 2 ml. of diluted acetic acid, and run a *U. S. P. heavy metals test:* the amount of heavy metals does not exceed 20 ppm.

Assay: (Salicylamide) Prepare a 0.001% solution of salicylamide as follows. Transfer to a 100 ml. volumetric flask 0.1 gm. of salicylamide, accurately weighed, fill to the mark with alcohol, and mix. Transfer to a second 100 ml. volumetric flask 10 ml. of this solution, fill to the mark with alcohol, and mix. Finally transfer to a third 100 ml. volumetric flask 10 ml. of this last solution, fill to the mark with alcohol, and mix. Spectrophotometrically determine the absorbancy in a 1 cm. quartz cell at 302 mμ, using alcohol as a blank. The concentration of salicylamide in the solution in mg./ml. = absorbancy ÷ 30.2. The amount of salicylamide is not less than 97 or more than 103%.

Prepare an approximately 0.1 *N* sodium methoxide solution as follows. Cut 3 gm. of freshly cut sodium in small pieces, and slowly add to 75 ml. of methanol. After all the sodium reacts, add an additional 75 ml. of methanol and 850 ml. of dry benzene, and mix. Standardize this solution thus. Transfer to a 100 ml. beaker, equipped with a magnetic stirrer and cardboard cover with a single hole for the buret tip, about 0.1 gm. of benzoic acid, accurately weighed, and add 30 ml. of freshly neutralized dimethylformamide containing a few drops of 0.3% solution of thymol blue in methanol. Add the sodium methoxide from a 10 ml. buret to a blue end-point. Store this standardized solution in borosilicate glassware protected against carbon dioxide and moisture. Transfer to a 100 ml. beaker, equipped with a magnetic stirrer and the same cardboard cover, about 0.1 gm. of salicylamide, accurately weighed, and add 30 ml. of freshly neutralized dimethylformamide containing a few drops of thymol blue. From a 10 ml. buret, add the freshly standardized 0.1 *N* sodium methoxide to the same blue end-point. Each milliliter of 0.1 *N* sodium methoxide is equivalent to 0.01371 gm. of salicylamide. The amount of salicylamide is not less than 98 or more than 102%.

Dosage Forms of Salicylamide

TABLETS. *Identity Tests:* Grind a number of tablets equivalent to about 1 gm. of salicylamide, and triturate the powder with three 25 ml. portions of ether. Filter the ether extracts through a pledget of cotton, and evaporate the filtrate to dryness on a steam bath. The residue responds to the identity tests for the active ingredient in the monograph for salicylamide.

Assay: (Salicylamide) Weigh 20 tablets, and powder them. Transfer to a 100 ml. beaker, equipped with a magnetic stirrer and cardboard cover with a single hole for the buret tip, an amount of powder, accurately weighed, equivalent to about 0.1 gm. of salicylamide. Proceed as directed in the titrimetric assay for the active ingredient in the monograph for salicylamide starting with, ". . . and add 30 ml. of freshly neutralized dimethylformamide . . . ." Each milliliter of 0.1 *N* sodium methoxide is equivalent to 0.01371 gm. of salicylamide. The amount of salicylamide is not less than 95 or more than 105% of the labeled amount.

The Bowman Bros. Drug Company, Canton, Ohio.

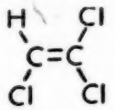
Hexett Tablets Salicylamide: 64 mg.

Tablets Salicylamide: 0.3 gm.

Chemo Puro Manufacturing Corporation, Long Island City, N. Y.

Powder Salicylamide: Bulk; for manufacturing use.

Trichloroethylene-U.S.P.—The structural formula of trichloroethylene may be represented as follows:



Actions and Uses.—Trichloroethylene is a volatile liquid that produces prompt analgesia and anesthesia when inhaled. Its action resembles that of chloroform but is more rapid and less potent. It is suitable for inhalation as an analgesic agent only. Anesthetic concentrations do not produce complete muscular relaxation and are associated with tachypnea and bradycardia, sometimes accompanied by extrasystoles. These are signs of overdosage. Tachypnea is a sign that the first plane of anesthesia has been reached and should be regarded as a warning that administration has exceeded the analgesic level. With a suitable inhaler, self-administration under professional supervision is considered relatively safe for producing analgesia, usually without unconsciousness. Patients may experience slight dizziness and numbness the first few minutes. Irritation of respiratory passages (excessive salivation or secretion of mucus), nausea, and vomiting are infrequent. If sufficient vapor is inhaled to produce unconsciousness, the mask automatically falls away from the face to prevent overdosage. Consciousness usually is restored within 20 to 30 seconds. Self-administration should not be permitted while the patient is alone.

Trichloroethylene as an analgesic mixture with air or oxygen is useful in obstetrics during labor and for delivery in conjunction with pudendal block or low spinal anesthesia. It also may be employed as an analgesic agent in minor surgical and

dental procedures and in major operative procedures as an adjunct to light general anesthesia produced by other agents. When this drug is administered with inhalation anesthetics, standard anesthetic machines with a closed circuit may be used, provided they are adjusted so that trichloroethylene does not come into contact with soda lime. If contact with soda lime occurs in closed apparatus, persistent trigeminal neuralgia and other cranial palsies may result from the formation of toxic degradation products (acetylene dichloride, phosgene, and carbon monoxide), formed by the interaction with soda lime.

Until further experience is gained, trichloroethylene is not recommended for use in patients with severe cardiac failure, active cardiac disease, or toxemia of pregnancy. It should never be employed for induction anesthesia. Administration of epinephrine should be avoided whenever trichloroethylene is used.

Dosage.—Trichloroethylene is administered by inhalation by means of an inhaler device controlled by the patient or a mask or closed-circuit anesthetic machine controlled by an anesthetist. Premedication can be carried out according to the preference of the physician. During labor or for minor surgical procedures, 10 to 12 self-administered inhalations from a suitable device are taken by the patient at the onset of pain. When this agent is dropped on a mask or placed in a machine, a minimal concentration should be maintained at all times to avoid even the first plane of anesthesia. Trichloroethylene is nonexplosive; it is not flammable when mixed with air, but may become so when mixed with oxygen. When the latter mixture is used, there is risk of ignition especially when a cautery is employed. In such instances, admixture with air is preferable. Trichloroethylene is highly stable when stored in closed containers away from light. However, to avoid possible oxidation it is inadvisable to retain any unused portion in an anesthetic machine; such portions should be discarded. Trichloroethylene may be heated without decomposition; but, when its vapor, diluted with air, is exposed to a naked flame, decomposition occurs giving rise to hydrochloric acid and traces of phosgene. The agent or its vapor should not be allowed to come in contact with hot surfaces.

Ayerst Laboratories, Inc., New York.

Trilene: 6 cc. ampuls and 30 cc. bottles. Stabilized with 0.01% thymol.

COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION

APPARATUS ACCEPTED

The following additional products have been accepted as conforming to the rules of the Council on Physical Medicine and Rehabilitation of the American Medical Association for inclusion in Apparatus Accepted. A copy of the rules on which the Council bases its action will be sent on application.

RALPH E. DE FOREST, M.D., *Secretary.*

Acousticon Hearing Aid, Model A-300

Acousticon Hearing Aid, Model A-300

Dictograph Products Inc., 95-25 149th St., Jamaica 35, Long Island, N. Y.

The Acousticon Hearing Aid, Model A-300, contains one transistor and two vacuum tubes and is powered by a mercury type A-battery and a 22.5 volt B-battery.

The body of the instrument measures 80 by 60 by 22 mm. and weighs 121 gm. With earphone, receiver cord, A-battery, and B-battery, the total weight is 165 gm.

Teca Portable Low Volt Generator, Model SP-2

Teca Corporation, 139 E. 23rd St., New York 10.

The Teca Portable Low Volt Generator, Model SP-2, provides a source of electric current for stimulating normally innervated muscle, for stimulating muscle with damaged nerve supply, for testing with respect to the reaction of degeneration, and for ion transfer. The output is either a continuous direct current or an alternating current. The latter consists of square waves at frequencies up to 2,000 per second; these can be amplitude-modulated at the rate of 6 to 60 surges per minute.

Teca Portable Low Volt Generator, Model SP-2

The apparatus is housed in a case with a carrying handle and a sloping front; it measures 26 (height) by 40 by 29 cm. (10 by 15½ by 11½ in.) and weighs 8.6 kg. (19 lb.). Packed for shipment it measures 30 by 45 by 35 cm. (12 by 18 by 14 in.) and weighs 10 kg. (22 lb.). It requires 60 cycle alternating current at 110 volts and draws 30 watts.

Beltone Hearing Aid, Concerto Model

Beltone Hearing Aid Company, 2900 W. 36th St., Chicago 32.

The Beltone Hearing Aid, Concerto Model, is a tubeless hearing aid that contains three transistors and requires one 1.25 volt mercury cell. The on-off switch and volume control are on one dial; the tone control is operated by a lever that has two settings.

The Concerto line includes: Green Dot, Yellow Dot, Red Dot, Black Dot, and Double Black Dot Models. Their characteristics are summarized as follows:

Beltone Hearing Aid, Concerto Model

Model	Battery				Power Output Sound-Pressure Level in Db. Above 0.0002 Dynes/Sq. Cm.
	Voltage	Capacity, Ma. Hr.	Drain, Ma.	Approximate Life, Hr.	
Green Dot.....	1.25	850	1.5	940	128
Yellow Dot.....	1.25	850	0.9	500	122
Red Dot.....	2.50	350	2.5	120	132
Black Dot.....	2.50	350	2.5	120	134
Double Black Dot.....	2.50	350	5.0	70	137

The body of the instrument measures 72 by 53 by 16 mm. and weighs 80 gm. The earphone weighs 8 gm., the receiver cord 2 gm., and the battery 12 gm., making the total weight 102 gm.

Sonotone Hearing Aid, Model 988

Sonotone Corporation, Box 200, Elmsford, N. Y.

The Sonotone Hearing Aid, Model 988, is an electrical instrument containing three vacuum tubes and powered by one 1.25 volt A-battery and one 15 volt B-battery. It has volume and tone control and receivers for both air and bone conduction. The body of the instrument measures 76 by 44 by 16 mm. and weighs 68 gm. The batteries, earphone and receiver cord weigh 24.3, 7.7, and 2.8 gm., respectively, making the total weight 103 gm.

Sonotone Hearing Aid, Model 988

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AIR POLLUTION

Air pollution is a complex problem that has been growing since coal was first used. Disasters due to the exposure of communities to dense smog for several days have focused public attention on the importance of control measures. In the latest such disaster, in December, 1952, in London, the concentration of smoke in the air reached 4.46 mg. per cubic meter, and that of sulfur dioxide reached 1.34 ppm.[1] Although heavy industries discharge vast quantities of smoke into the air, coal burning railroad engines, motor vehicles, and furnaces from private homes and apartment buildings are also important sources of air pollution.

While most persons recognize air pollution as a nuisance, the determination of concentrations of various pollutants necessary to be definitely injurious to health is a far more difficult matter. The chief effects noted are irritation of the upper respiratory tract and conjunctiva. In this problem the effects of exposure to high concentrations of pollutants for a short period and the effects of prolonged exposure to low concentration are usually studied. The latter is especially difficult to evaluate. The maximum allowable concentrations of various pollutants in a community can best be determined by studying their effects on a more limited scale in industrial workers subject to daily exposures to polluted air.[2] In order to establish a causal relationship between disease and exposure it is necessary to show that "(1) a toxic effect has occurred; (2) a pollutant capable of causing this effect is (has been) present; (3) there is (has been) an excessive exposure to the pollutant; and (4) other causes of the toxic effects do not exist."[3]

In attempting to get remedial measures adopted it is well to reinforce hygenic arguments with other, more purely economic, arguments, such as toxic effects on grazing animals in some areas; reduction in agricultural yield; discoloration and disintegration of stone, mortar, and metals of buildings; increased cleaning and illumination costs; and loss through dispersion of valuable by-products.[1] Residential districts should not be built in the direct line of the prevailing wind from a heavy industry. When the wind is found to blow heavy smoke from a plant toward such sections or when atmospheric conditions favor smog, extra controls in the form of filtration or partial shutdown should be applied. Industries should especially avoid locating in narrow valleys subject to frequent stagnation of the air by having meteorological surveys made of proposed sites.

That something can be done about air pollution has been amply demonstrated in Detroit,[4] Los Angeles,[5] and other cities. In 1947 Detroit organized a control program. As a result, the average horizonal visibility has been greatly improved and fly ash no longer accumulates along curbs and in doorways. In Los Angeles it is estimated that over 600 tons of air pollutants per day formerly dispersed over the city are now withheld from the air; 360 tons of sulfur and 100 tons of gasoline are reclaimed daily; the sulfur dioxide content of the air has been reduced 50%; and the average visibility has been increased. These improvements have resulted from a large outlay in time and money by the community and industries, but they have been worth the cost.

Increased populations, technological changes in industry, and the use of new products give rise to new community problems faster than the old ones can be solved. No complete solution to the problem of air pollution can be expected, but there is a great need for those cities that have not yet done so to clean up their air. Other cities have shown that it can be done.

COLD INJURY

Although military operations are attended by the greatest incidence of cold injury, many persons in civil life may be suddenly exposed to frostbite or immersion foot as a result of a cyclone, flood, or occupational hazard.[1] The danger is increased if the person exposed has an appreciable degree of peripheral arteriosclerosis. In the production of tissue damage, it was formerly believed that the associated vascular changes were secondary to the cold injury, but it now appears that the tissue damage is secondary to such vascular changes as vasoconstriction, vasodilation, sludging of blood, and thrombosis. The feet are especially vulnerable because of their dependent position. The damage may be aggravated by immobility, which favors sludging of the blood, as well as by walking. That the exact pathogenesis of cold injury is complex and not fully understood is shown by the fact that some animals can hibernate without incurring cold injury and certain tissues can be refrigerated and later used as grafts.

There is a pronounced difference in the ability of different persons to withstand exposure to cold and in the same person at different times. In addition to the temperature and duration of the exposure, the humidity and wind velocity enter into the picture. Advanced age, especially in the presence of peripheral vascular disease; poor general physical condition; high cold agglutinin titer; belonging to a dark skinned race; and having had a previous frostbite decrease resistance to cold injury and should be taken into account in any effort to prevent such injury.[2] It is estimated that only about 50% of

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1. Pratt, G. H.: Frostbite Therapy, J. Am. Geriatrics Soc. **2**:305-316 (May) 1954.

those who incur cold injury had warning symptoms of sufficient severity to attract attention. These patients may complain of feeling cold, stinging pain, and numbness in the involved part. Some of these patients, believing their feet to be asleep, pound them or hop on them "to get the circulation started." This trauma increases the ultimate tissue damage. Unfortunately, others remove their shoes to obtain relief and then because of swelling are unable to get them on again, so that greater exposure occurs. Exposure to cold in high altitude flying is accompanied by anoxia. This also aggravates the injury.

If the circulation is not irreparably damaged the tissues will regenerate very well, but clinical evaluation of any form of treatment is made difficult by the fact that examination of the recently injured part at the time treatment is started gives no clue of the extent of the injury. For this reason Isaacson and Harrell [3] studied 33 patients who were believed to have had bilaterally equal injury. They performed a unilateral sympathectomy so that each patient was his own control. The aim of treatment is to prevent gangrene, but in their series there was no significant difference in the extent of the gangrene or amputation required between the treated and the untreated side. They concluded that sympathectomy is useful only in the treatment of late sequelae (healing chronic ulcers, relieving paresthesias, or improving circulation).

In the emergency phase of treatment there was some difference of opinion as to the relative merits of slow rewarming and rapid rewarming. As the result of animal experimentation rapid rewarming is now generally accepted as the method that will result in the least gangrene, but there are still some differences in the details of the technique advised. Because water is a better conductor than air, rapid rewarming should be accomplished in a water bath. Russian investigators [4] warned against using bath temperatures higher than 98.6 F, but Gottschalk recommends a temperature of 108 F.[5] Pruitt [6] recommends a temperature of 86 to 89 F and warns against temperatures of 100 F or over. A more accurate determination of the optimal temperature of the rewarming bath could be arrived at by using a controlled series such as that reported by Isaacson and Harrell. All agree that massage should be avoided. Once the part has been rewarmed it should be left exposed in cool surrounding air at 70 to 78 F. Pressure dressings and the application of ointments are to be avoided. Because smoking causes vasoconstriction it should also be avoided.

Some authors favor the use of heparin to prevent thrombosis, provided there is no evidence of bleeding, but others believe that it is of doubtful value and that it adds an unnecessary risk. If it is used, it should be given early in the course of treatment.[5] Vasodilators have not been shown to be of value, except possibly in patients who show signs of sympathetic overactivity during recovery. Corticotropin, cortisone, and rutin have not been found to have any favorable effect. Pratt [1] advocates the use of procaine intravenously for pain, but Gottschalk states that such drugs as meperidine are safer and easier to administer. Bed rest is necessary to prevent trauma until the bullas have dried. The surgical treatment should be ultraconservative because when the necrotic tissue has sloughed off there is usually much less loss of tissue than was anticipated. Some grafting may be required, but amputation should be used only when spreading infection endangers the patient's life.

Great strides in our understanding of cold injury have been made since the days when such injuries were rubbed with snow. Recent military campaigns have provided ample material for observation and have helped to clarify some of the points of major controversy.

RENAL TRAUMA IN PROFESSIONAL BOXERS

Physicians who are attracted to the numerous telecasts of boxing prowess that are indigenous to the American scene may have wondered about the severity and extent of renal injury sustained by professional boxers. An answer is partly supplied by a recent study by Amelar and Solomon [1] on 103 professional boxers who fought at the Madison Square Garden and at St. Nicholas Arena in New York City during 1952 and 1953. A freshly voided specimen of urine was collected from each boxer on the day of the fight and examined within an hour. After a bout, another specimen was obtained from each boxer within 5 to 20 minutes. Each specimen was examined for color, turbidity, specific gravity, pH, glucose, albumin, and acetone; a detailed microscopic examination of the sediment was also made. In 46% of the fighters the urine, which was clear before a bout, became turbid afterward. Albumin, not present before the bout, appeared in 68% of the urinary specimens after the pugilistic encounters. Traces of acetone appeared in the urine of 14% of the boxers, urinary specific gravity increased in 80%, urinary pH decreased in 39%, and glucose appeared in 9%. Significantly, microscopic examination showed that in 73% of the urine specimens red blood cells appeared immediately after a bout, while 26% of the boxers had granular or hyaline casts in the postcombat urine. According to the authors, red blood cells and casts are not found in the urine of persons engaging in strenuous exercise, although various investigators have reported that exercise produces a decrease in urine volume, a reduction in renal plasma flow and filtration rate, a decrease in the chloride concentration, and an increase in urinary acidity, ammonia, and phosphates. Strenuous exercise can cause albuminuria.

From this study it appears that the effects of acute renal trauma sustained in professional pugilistic bouts can be determined in part by appropriate urinalysis. Acute renal trauma is revealed by the findings of red blood cells, casts, and a high percentage of albumin. The door remains wide open for further study of other aspects of the problem, such as the question of the effect of repeated injury to the kidneys on renal function. This might be done on a group of boxers who are retired. An immediate application of this study is the suggestion that a boxer who has sustained renal punishment should not be allowed to fight until his urinary findings are normal.

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ORGANIZATION SECTION

CIVIL DEFENSE

Following is the first section of the third and last of a series of articles on medical civil defense plans that have been prepared for the state of Maine. These plans are being published as samples for those states that have not as yet completed this phase of medical civil defense planning.

Plans for Special Weapons Defense. Part III: Chemical Agents Defense Plan, Section 1

Adequate measures for safeguarding the civil population against the direct effects of modern war must become an essential part of any state civil defense organization if the nation is to survive in this age of fast, long-range jet bombers and supersonic guided missiles. It is just as necessary that these preparations for civil defense be made in time of peace as it is that the armed forces be maintained in a state of readiness at all times. Furthermore, any up-to-date, well-balanced plan for effective civil defense must take into account all the various weapons that might be available to an enemy. It is imperative that a proper balance be held between the likely and the possible special weapons. Civil defense agencies must be prepared to deal immediately with all probable types of attack but should be especially alert to those types that could inflict heavy damage to life and property from a long range. Among these are attacks by air with the newer agents of chemical warfare like the nerve gases or, possibly, with mixtures of the older types of vesicants and the newer nerve poisons. The Germans had a large quantity of the nerve gas "Tabun" on hand at the end of World War II that they had never used against the allied armies or their civilian population centers. The Russians are known to have captured several hundred tons of nerve gas and to have taken over the German nerve gas production centers and most of the scientists who were familiar with the manufacture of these new chemical agents. Therefore, it must be assumed they know as much about this new group of chemical weapons and their potentialities as does our own Armed Forces Special Weapons Division. Neither the United States of America nor the Union of Soviet Socialist Republics was a signer of the Geneva Gas Protocol of 1925; hence, the United States has at present no signed agreement with any nation guaranteeing that chemical warfare agents will not be used against us in the event of war. The fact that gas was not used during the last war might well increase the possibility of its future use in the hope of achieving the surprise on which the success of any gas attack largely depends. Large bomber and fighter airfields have been built and activated within this state beside one of our largest cities and two of our medium-sized towns. Any attack on these military targets with nerve gas could subject much of our civil population to the lethal effects of these new chemical agents, if the wind were blowing from the airfield toward the urban center. Furthermore, nerve gas bombs might be jettisoned onto other cities during an enemy air raid if their bombers were closely pursued by our fighters. Hence, the Maine civil defense authorities are agreed that gas warfare is still one of the risks that the civil population of this state must be immediately prepared to face and that chemical defense training must be instigated and provisions made for providing protection, equipment, and supplies for use by the detection and decontamination units, as well as for the civil population in certain urban centers.

The chemical defense organizational and operational plan prepared for Maine closely follows that devised by the armed forces of the United States since World War II. It will be presented in two sections. Organization and operation of detection units and specific detection procedures are outlined below; the organization and operation of decontamination units and specific decontamination procedures will comprise the second section of this chemical defense plan. Any plan for defense against chemical warfare agents may have to be altered from time to time; hence, only basic organizational and operational procedures are outlined in essential detail, and these will need to be supplemented as additional material and information become available.

RECOMMENDED TABLE OF ORGANIZATION AND EQUIPMENT

Chief of Section.—An Assistant State Deputy (Chemical) Director #3 to serve as "Chief," Chemical Defense Section, whose duties will be as follows.

1. Responsibility through the "Chief," Special Weapons Division, to the State Deputy Director #3 for (*a*) organization of a State Advisory Panel and presiding at the meetings; (*b*) preparation of a State Chemical Defense Plan, after consultations with the State Advisory Panel and the Chief of the Special Weapons Defense Division; (*c*) organization and training of the required number of high-level chemical detection units to be located at the state university, the three Maine colleges, and strategically located, large industrial plants within the state; (*d*) preparation of a questionnaire listing dangerous chemicals, to be sent to all plants in the state to determine the number and quantities of such hazardous chemicals on hand at each mill or plant; (*e*) preparation of a set of field and high-level laboratory tests for the identification of chemical warfare agents, after consultations with the State Advisory Panel and the Chief of the Special Weapons Division; (*f*) organization and training of special weapons decontamination units as required at strategically located industrial plants and mills throughout the state, after consultations with the chiefs of the Radiological and Biological Sections and the Chief of the Special Weapons Division; and (*g*) preparation of the specific chemical decontamination procedures to be carried out by the special weapons decontamination units.

2. Advising each county civil defense director and his #3 deputy director and the chief of the latter's chemical defense section in regard to the number and location of chemical detection and special weapons decontamination units required in said county.

3. Prescribing the chemical defense training schedule to be followed by detection and decontamination units organized at the regional, county, or local levels.

4. Working closely with the chiefs of the Biological and Radiological Sections and the Chief of the Special Weapons Division with a view to working out a well-coordinated and carefully integrated special weapons defense plan and organization for the entire state.

State Advisory Panel.—This group will:

1. Consist of professors of chemistry of college or graduate-school level and selected research and analytical chemists from large industrial plants.

2. Operate under the direction of the Chief of the Chemical Section.

3. Assist the Chief of the Chemical Section in working out chemical agent identification and decontamination procedures and techniques to be used by the chemical detection and decontamination units.

4. Help establish at strategic locations, preferably at the colleges or in the industrial laboratories, facilities for the identification of unknown chemical agents that might be used against our military targets and industrial centers in war.

5. Assist with the organization and training of the regional chemical detection units described below. The members of the panel may, if they choose, join these units for training and during emergency operations. Furthermore, members should assist the county and local chemical defense chiefs with the training of lower-level detection and decontamination units organized within their respective regions.

Chemical Agents Detectio.. and Reconnaissance Units.—These units are organized as follows.

Senior Chemical Officer—State and Regional: Professors of chemistry from the university and from each college have been

designated to direct the formation of chemical detection units in the vicinity of their respective schools and to assist in the training of the personnel assigned to each such unit. The senior chemical officer and the members of his unit will be under the direction of and responsible to the Assistant (Chemical) State Deputy Civil Defense Director #3. Research or analytical chemists from large mills or industrial plants have been designated to organize chemical detection units and decontamination units in their respective mills or industrial plants. These unit leaders will serve as regional senior chemical officers and will be under the direction of and responsible to the Assistant (Chemical) State Deputy Civil Defense Director #3. These regional chemical agent detection and reconnaissance units may be attached to the nearest state mobile battalion for transportation, messing, and billeting during actual operations in an emergency certified by the governor of the state.

Senior Chemical Officer—County or City: An instructor in chemistry or an industrial chemist should organize and train chemical and bacteriological detection units at the county and town levels where such units are required. This officer should be responsible to the County or City Deputy Director #3 for developing and promoting the chemical and bacteriological detection program at these lower levels.

The composition recommended for chemical and bacteriological identification and reconnaissance units is as follows.

State and Regional Units Under State Direction

Chemist (1)		Chemists (2)
Bacteriologist (1)		Chemical assistants (2)
Chemical assistants (2)	OR	Decontaminator (1)
Decontaminator (1)		Driver-loader (1)
Driver-loader (1)		

County and City Units Under Local Direction

Chemist (1)		Chemists (2)
Chemical assistant (1)		Chemical assistants (2)
Bacteriologist (1)		Decontaminator (1)
Bacteriological assistant (1)	OR	Driver-helper-loader (1)
Decontaminator (1)		
Driver-helper-loader (1)		

Duties of the Field Detection Units.—The first duty of field detection units after an enemy attack should be to confirm the presence or absence of war gases in suspected areas and to report the type of gas used and the extent of the contaminated areas to the control authority as soon as possible. Wind direction and the rate of spread of vapor gases should also be determined and Civil Defense Headquarters informed. All contaminated areas must be marked off. Persons in contaminated areas at the time of the attack must be brought out of the gassed area as quickly as possible, first aid procedures instigated promptly, and the badly gassed persons evacuated to medical installations for treatment. All persons must be evacuated from the downwind section adjacent to gassed areas. Unauthorized persons must be kept out of the danger areas until suitable decontamination procedures can be completed.

Field detection teams must provide the high-level detection units with samples of air containing the vapor form of unknown war gases. A description of the apparatus and the techniques to be used in collecting contaminated specimens appears below. The field detection units must collect samples of contaminated solid or liquid materials to be sent back to the high-level units for more detailed testing and analysis.

Methods and Equipment for Detection Units.—A chemical agent sampling kit will be needed that should contain several empty screw-cap sample bottles, a scoop, long medicine droppers, rubber gloves, blotting paper, cotton toweling, and a notebook and pencil. The apparatus contained in this kit will enable the unit in the field to take samples of solid chemical agents suspected of being in the soil, on vegetation, or on hard surfaces. Each detection unit should carry one combustible gas indicator, similar to that made by the Davis Emergency Equipment Co., Inc., Newark, N. J., for the detection of inflammable gas mixtures. This equipment is necessary for the protection of unit personnel when they are obliged to enter areas where gasoline, benzol, turpentine, etc., may have been liberated or buildings where gas mains may have been broken, releasing city gas or commercial gas in explosive concentration.

The members of the gas detection units should always approach every area suspected of gas contamination with great care and with all senses alert. It is one of the most fundamental duties of all concerned with detection and reconnaissance not to get themselves contaminated, or become casualties, through carelessness. If they do, they are unable to carry out their particular task and, at the same time, will put a still further load on the medical services. Detection personnel should try to keep upwind of contaminated areas except for that actually being investigated at the moment. A continuous watch must be kept on the direction of the wind in the vicinity, and the position of any other gas incidents previously reported should be borne in mind. The nonpersistent gases or particle clouds will be difficult to collect in most instances because this vapor or cloud will probably be dissipated before the field detection unit can reach the gassed area. However, an attempt can be made to collect samples by placing unimpregnated silica gel granule detector tubes in either the Andrews (fig. 1) or the Bournique (fig. 2) gas detection apparatus and drawing specified quantities of the contaminated air through the collection tubes. Vapor from an agent of low volatility can be concentrated by placing a can or box over the liquid for a few minutes, which allows time for sufficient vapor to collect inside the container. Then a small hole should be made in the container, and the funnel at the distal end of the collecting tube attached to the collecting apparatus should be placed over the hole and the contaminated air drawn up through the silica gel collecting ampul. Samples of air suspected of containing war gas vapor should always be taken on the downwind side but close to the source of contamination, such as a shell hole or an object suspected of being contaminated.

CHEMICAL AGENTS DETECTION PROCEDURES

The development of the extremely potent new group of almost odorless and colorless nerve gases—Tabun, Sarin, and Soman—by the German chemists prior to and during World War II has rendered obsolete many of the older war gases and has necessitated a radical revision in recommended detection methods. Chemical tests for the war gases must be relied on instead of the senses of smell, sight, and touch. Great stress must also be placed on keeping a careful watch for the sudden appearance of any of the following symptoms among persons out in the open immediately after any possible enemy attack: (1) pinpoint pupils with difficulty in focusing on near objects, (2) acute frontal headache, (3) running nose, (4) an asthmatic feeling of choking and tightness in the chest accompanied by cyanosis and severe bronchial spasm that obstructs both inhalation and exhalation, (5) nausea and vomiting, (6) mental confusion and loss of consciousness, and (7) convulsions and death. These clinical effects on exposed persons will likely be the first and best indication that nerve gas has been released in the area, since no simple, quick chemical test for the satisfactory initial detection of nerve gas has yet been made known. However, reliable chemical detection tests and practical detection sets will be required for detecting persistent vesicant and nerve gases in liquid form and for determining when the volatile forms have become sufficiently dissipated by the wind to permit personnel to enter areas without gas masks and protective clothing. Until our enemies establish military bases on the North American continent, it would seem that civil defense against chemical attack could, for practical purposes, be reduced to detection methods for nerve gases; however, it should be kept in mind that some persistent vesicant gas could be used with volatile nerve gas to hamper rescue operations, or mixtures of war gases might be used to make identification more difficult. Of the war gases now known, use of the nerve gases Sarin and Soman and the vesicants mustard and nitrogen mustard is considered most probable. Lewisite, dichloroethylarsine, hydrogen cyanide, phosgene, and cyanogen chloride are the likelier of the other war gases for which high-level detection units should have identification tests, in case certain of these chemical agents should be mixed with nerve gases for their nuisance value.

Field detection teams will be required to wear the gas mask and impermeable, butyl-rubber-coated protective clothing consisting of a hood, a coverall-type, single-piece suit, gloves, one pair of boot covers, and one pair of rubber boots until the presence of persistent liquid nerve gas can be ruled out. Since the impermeable suits and accessories are impervious to air and to body perspiration, the suits will very quickly give rise to great discomfort to personnel wearing them during hot, humid weather unless a two-piece, diaper-cloth suit is worn over the impermeable suit and kept well wet with water. Obviously, chemical detection personnel with a limited amount of technical training and skill and wearing heavy-duty gas masks, heavy rubber gloves, and other protective clothing cannot be expected to carry out a series of complicated detection tests for poisonous gases in the field under wartime disaster conditions. The field detection and collection apparatus should be easy to operate, and the field detection tests should be single-step, easily carried out procedures limited, if at all feasible, to one or two in number.

Detection of War Gases by Field Units.—Mr. A. B. Andrews, Chief of the Special Weapons Division, has designed a simple apparatus (fig. 1) for the identification of war gases in the field

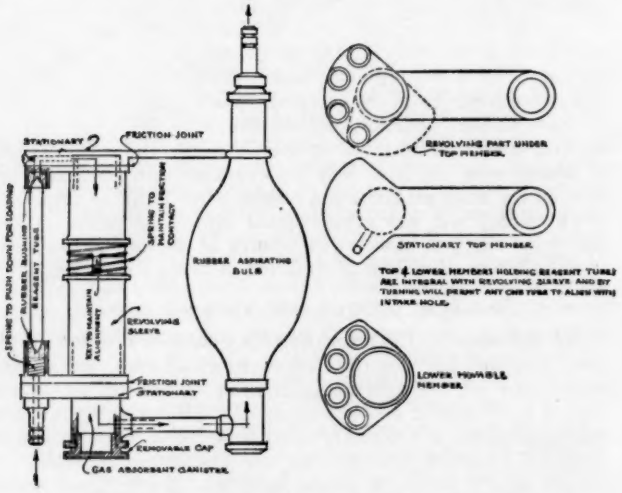

Fig. 1.—Andrews apparatus for field testing of war gases.

and for the collection, on silica gel granules in ampuls, of the air samples required for the high-level tests. This apparatus is a modification of an apparatus originally designed by the Mine Safety Appliances Co. for the detection of carbon monoxide. The direction of flow has been reversed and an intake and outgo manifold added so that one or two ampuls containing test reagent on indicator paper or silica gel granules and two unimpregnated gas collection ampuls containing plain silica gel granules can be inserted into the apparatus at the same time. The contaminated air can pass simultaneously through the detection and collection ampuls first, after which it will pass through a gas-absorbent canister before reaching the inside of the suction bulb; or air can be drawn through test ampuls one at a time by simply closing a cutoff to each of the other test and collecting ampuls.

Universal Indicator Paper or Solution as a Qualitative Test for Probable Hydrolyzable War Gases: Universal indicator may well prove to be a good single, qualitative test reagent for the detection of war gases in the field. Nitrogen mustard gives an alkaline reaction, whereas all of the other more probable vesicant gases, choking gases, and blood gases and, presumably, Taban, Sarin, and Soman give an acid reaction with the universal indicator. It is believed that universal indicator paper, with proper moisture content and suitably sterilized, could be sealed into glass ampuls designed for use in the Andrews detection apparatus. An acid or an alkaline reaction on the indicator paper would indicate the possible presence of war gases in an area. Detection personnel should then collect samples of the contaminated air on silica gel granules for more specific testing by the high-level detection personnel outside the area of contamination. Other nonwar gases in the area might give false positive tests with universal indicator, but it would be equally unwise for persons to enter areas where such gases were in sufficient concentration to give a reaction with universal indicator test paper. It will be necessary to ascertain the effects of the various chemical agents in the gaseous state on universal indicator paper before it will be known with certainty whether all the gases named above will give either an acid or an alkaline color reaction at sufficiently low concentrations. If some or all of the nerve gases fail to give a distinctive reaction in the vapor form with universal indicator paper, it will be necessary to devise some other simple qualitative test for the detection of all types of war gases in the field; if a single, simple test cannot be perfected, separate tests for the identification of the nerve and the vesicant agents in the gaseous state may be required. Examples of such diagnostic tests that may prove adaptable for field use are listed below.

Silver Test for the Detection of Nerve Gases: The nerve gases Sarin and Soman are basically phosphine oxide derivations. Since phosphine may be detected by the brown to black color

Materials Required for Detection Units

Equipment	No. Required Per Unit
1. Chemical agents detection kit, field type.....	2
One of the following types should be selected	
(*a*) Andrews (bulb type)	
(*b*) Lever air flow type	
(*c*) Bournique type	
(*d*) Jacobs type	
2. Chemical agents detection kit, high level type *.....	2
One of the following types should be selected	
(*a*) Lever air flow type	
(*b*) Bournique type	
(*c*) Chemical Agent Detector Kit M9-A2,† Department of the Army, if available	
3. Combustible gas indicator.....	1
Supplies	
1. Reagents required for detection apparatus selected	
2. Replacement glassware and tubing for detection apparatus used	
3. Supplies required for adequate decontamination of detection apparatus and protective clothing ‡	
4. Marker signs and marker tape for marking gas-contaminated areas	
Impermeable Protective Clothing (Butyl Rubber Coated)	
1. Suit, protective, impermeable, one piece, M3 †.....	8
2. Hood, protective, impermeable, M3 †.....	8
3. Gloves, protective, impermeable, M3 †.....	8
4. Boots, rubber, short, heavy knee, pair.....	8
5. Covers, boot, impermeable, pair, M1 †.....	8
6. Suit, cooling, two piece (jacket & trousers) diaper cloth, M1 †	8
7. Hood cover, cooling, M1 †.....	8
8. Mask, protective, M9A1 † or other approved type.....	8
9. Impermeable protective apron, M1 †.....	8
Protective Accessories	
1. Protective ointment kit, M5A1 †: includes.....	8
(*a*) Protective ointment, 3 tubes, M5 †	
(*b*) BAL eye ointment, 1 tube	
(*c*) Atropine tartrate injection, 3 tubes	
2. Protective dubbing for shoes or leather boots, can.....	8
Decontamination Equipment	
1. Decontamination apparatus, 1½ qt., M2 †.....	4
2. Decontamination apparatus, 3 gal., M1 †	

* High-level detection kits should be supplied only to regional units under state civil defense control, and not to county and city units under local control.

† The letters and numbers refer to the armed forces numbers for these items and are included to facilitate matters if these items are eventually made available to civil defense agencies.

‡ To be described in Section 2 of this plan, to be published.

imparted to ammoniacal silver nitrate solution, it may be possible to detect nerve gas vapor in the field by drawing air through an ampul that contains silica gel previously impregnated with ammoniacal silver nitrate solution. The silica gel should darken and finally assume a brownish color if nerve gas is present in the air sample. Actual experiments using the nerve gases in vapor form will be necessary to determine whether a distinctly positive test will be given with this ammoniacal silver nitrate reagent under conditions that would exist in the field. All ampuls containing this silver test reagent would need to be wrapped in tinfoil or dark paper to keep out the light prior to their actual use. Heat might have to be applied to the test ampul after the air sample is passed through the silica gel impregnated with the ammoniacal silver nitrate solution in order to induce a positive test reaction. Any acid gas in sufficient concentration would neutralize enough of the base to permit the appearance of a colored silver compound on the impregnated silica gel.

Acidified Iodoplatinate Paper for the Detection of Nitrogen Mustard Vapor: Nitrogen and sulfur mustards are the two next most probable to be encountered of the war gases. Nitrogen mustards are bases and will give a basic reaction with suitable indicators. A more specific test for both vapor and liquid forms of nitrogen mustards is acidified iodoplatinate paper. This indicator paper can be made by soaking filter paper in a solution containing sodium iodoplatinate, glycerol, and phosphoric acid and then drying in a clean atmosphere. This acidified iodoplatinate indicator paper should turn blue when air containing nitrogen mustard gases, either as free bases or in the form of salts, is drawn through an ampul containing the indicator paper in the Andrews apparatus.

Congo Red Indicator Paper Test for Mustard Gas Vapor: The mustard gases give an acid reaction with suitable indicators and, in this respect, differ from the nitrogen mustards, which give an alkaline reaction. The Congo red indicator test for mustard gas depends on the reaction of a stable chloroamide with mustard gas with the liberation of free hydrochloric acid, which turns Congo red indicator paper blue. Halozone might meet the requirements for a stable chloroamide, but this chloroamide would need to be tried out in tests with the mustard gases. The air to be tested (either atmospheric or that which has been contaminated by passage through earth samples) is drawn by means of the bulb of the Andrews detection apparatus through an ampul containing Congo red paper that has previously been spotted with a solution of the stable chloroamide dissolved in carbon tetrachloride and allowed to dry. When mustard gas vapor in high concentration is present in the air sample, the spot darkens and finally appears as a blue area on a red background. Nitrogen mustard gives a negative reaction with Congo red test paper spotted with a saturated carbon tetrachloride solution of a stable chloroamide. This test has the added advantage that the amount of aspirated air required to produce this change can be used as an indication of the concentration of the mustard gas. Acidic vapors and all strong acids interfere with this Congo red-chloroamide test for mustard gases, but this situation can be utilized in the detection of easily hydrolyzable chemical agents such as lewisite in earth samples. The presence of such poisonous substances should be suspected if all the Congo red paper turns blue. The chemical warfare agents that will cause the overall blue coloration of the Congo red test paper are lewisite, methyldichloroarsine, phenyldichloroarsine, cyanogen chloride, sulfur trioxide, chlorosulfonic acid, and white phosphorus. When both acid gases and mustard gas are suspected, the former may be removed by passing the air sample through a filter that has been impregnated with a 10% solution of sodium carbonate and allowed to dry. The mustard gas vapor passes through the sodium carbonate filter and reacts in the usual way with the chloroamide spotted on the Congo red paper.

Detection of the Persistent Liquid War Gases in the Field.—Each field detection unit should be equipped with a shaker package containing an indicator powder that will give a distinctive color change when brought in contact with persistent vesicant and nerve gases in liquid form.

Detector Powder MK.1: The MK.1 powder, devised by the British, comes in shaker packages suitable for use by field detection teams. The following instructions appear on the MK.1 pack. "Sprinkle a thin layer of powder over suspected damp surfaces. The rapid appearance of a bright color denotes presence of liquid war gas. Nerve gas gives a yellow or orange color. Blister gas gives a reddish or purple color." This powder is ideal for the detection of persistent war gases in the liquid form, since it is claimed that the powder gives these distinctive color changes for both the nerve gases and the blister gases. Inquiries have been made through Mr. D. C. Evans, A.D., Tech Services (Land), British Joint Services Mission, Washington, D. C., with a view to arranging the purchase of a supply of this powder through regular commercial channels.

The Lever Detector Powder and Crayon for the Detection of Liquid Blister Gases: Mr. Wendall Lever, former chief of the Maine Civil Defense Chemical Section, has devised a detector powder for use by the field detection units in testing for persistent liquid blister gases. This powder is made up of 1 part of Sudan IV, 5 parts of Sudan III, and 94 parts of talc or chalk. A blue-green color develops as soon as the powder comes in contact with any of the liquid vesicant gases. Persistent nerve gases do not, as far as is now known, react with this Lever indicator powder. An indicator crayon can be made up from 1 part of an equal mixture of Sudan III and Sudan IV, 9 parts of plaster of Paris, and 90 parts of precipitated chalk. The ingredients are mixed and enough water added to make a thick paste, which is transferred to molds and allowed to dry. This Lever crayon shows an immediate blue-green color when the persistent blister gases come in contact with the Sudan III and IV dyes in the detector crayon.

The S. D. Warren Company Indicator Paper for Detection of Blister Gases: An excellent white vesicant-indicator paper was developed by the S. D. Warren Co., Inc., Westbrook, Maine, for the State Civil Defense Agency in Maine, Massachusetts, and Rhode Island during World War II. This indicator paper turns bright blue when the persistent liquid vesicants come in contact with it, but as far as is known this paper will not indicate nerve gas in any form. This vesicant indicator paper can still be purchased from the manufacturer.

Confirmatory Tests for the Specific Identification of War Gases.—Since the introduction of chemical warfare during World War I, many new agents have been developed as chemical weapons. Information about many of these substances has been carefully restricted; hence, it is difficult to propose a scheme of detection for chemical warfare agents that will be entirely adequate. As a further result of this secrecy we must largely rely for detection on techniques developed from 1918 to 1942, supplementing these with tests based on such meager information as is available concerning the more recently developed war agents. It is on this basis that the following scheme of analysis is proposed for use by the Maine Civil Defense Agency. The information that follows has been gathered from a number of unrestricted sources, few of which have originated since 1943.[1]

The various confirmatory tests for the detection of chemical warfare agents can be classified into two groups, namely field tests (F), those that are simple and give an immediate, detectable response to the agent and are, therefore, suitable for field use for immediate warning of the presence of gas, and high-level tests (H), those more complicated ones that do not permit an immediate detection, since one or more chemical operations

1. Sources of the information in this article and other publications on this subject are: Sartori, M.: The War Gases: Chemistry and Analysis, New York, D. Van Nostrand Company, Inc., 1939. Jacobs, M. B.: War Gases: Their Identification and Decontamination, New York, Interscience Publishers, Inc., 1942. Studinger, J.: A Short System of Analysis for the Detection of Materials Used in Chemical Warfare, Chem. & Ind. **56**: 225, 1937. Hoogeveen, A. P. J.: The Detection of Small Quantities of War Gases, ibid. **59**: 500-556, 1940. Hickey, F. C., and Hanley, J. J.: War Gas Identification Sets, J. Chem. Educ. **19**: 360, 1942. Bradley, T. F.: Chemical Detection of War Gases for Civilian Defense, Chem. & Engng. News **20**: 893, 1942. Identification of Chemical Warfare Agents, Publication 2219, United States Office of Civilian Defense, Medical Division, October, 1943. Snell, F. D., and Snell, C. T.: Colorimetric Methods of Analysis, ed. 3, New York, D. Van Nostrand Company, Inc., 1949, vol. 2, pp. 660-672. Jacobs, M. B.: New York City Department of Health Chemical Agent Detector Kit, issued by the author, January, 1954. Chapman, N. B.; Heap, R., and Saunders, B. C.: Determination of Fluorine in Organic Compounds, Analyst **73**: 434, 1948. McCombie, H., and Saunders, B.: Alkyl Fluorophosphonates, Nature **157**: 287 and 776, 1946; Fluoroacetates and Allied Compounds, ibid. **158**: 382, 1946. Sartori, M. F.: New Developments in the Chemistry of War Gases, Chem. Rev. **48**: 225, 1951.

are involved after the sample has been collected, and are, therefore, best done in the laboratory. Tests of type F can generally be carried out with simple equipment by personnel that has had a minimum of chemical training. However, such tests are rarely specific for any one war agent and often give false positive tests; therefore, it is necessary to supplement such tests with those of type H, which usually require somewhat more elaborate equipment and more thoroughly trained personnel, but which can often give very specific information concerning the nature of the war agent. In the following discussion each test will be classified as either F, suitable for field use, or H, more suitable for high-level laboratory use. The F tests will be given first in each of the sections listed below.

Some of the F tests can be carried out by the use of test papers or powders that are sensitive to the war agent in question and undergo a change of color on exposure to contaminated air, liquids, or surfaces. In some cases it may be more convenient to use an aspiration system, which draws suspected air through or over an active form of the test material on test paper or silica gel granules. When this is the case, the apparatus shown in figure 1 or figure 2 may be used. The apparatus shown in either figure 3 or figure 4 may generally be used for H tests. The Lever air-flow test equipment (fig. 3) uses the aspirator principle with an intake and outflow manifold assembly. A set of five test tubes measuring 1 by 4 in. (2.54 by 10.16 cm.), mounted in easily accessible frames, should suffice for field detection use; about 10 test tubes would be more suitable for use by the high-level detection units. Each tube is fitted with a three-hole, plastic stopper. A glass tubing manifold input system is piped through one hole in the stopper to the bottom

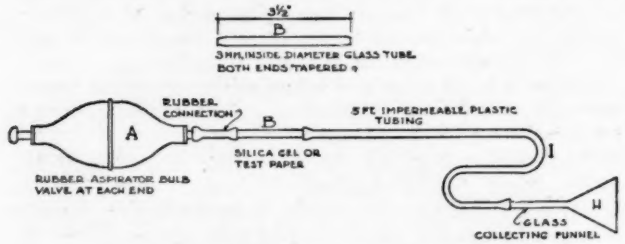

Fig. 2.—Bournique reaction-tube apparatus.

of each tube to permit the incoming gas to bubble through the test solution. The output glass tubing manifold is piped from each active tube through the second hole in the stopper with the takeoff end in each tube about ½ in. (1.27 cm.) below the stopper. A short piece of plastic tubing is attached to the outside end of a piece of glass tubing that goes through the third hole in the stopper; a glass rod is used to plug the open end of this plastic tube. This glass rod is easily removed, and any additional reagent required to complete a reaction is added through the open end of the plastic tubing. All flexible tubing in this Lever apparatus should be made of Saran or Tygon. A large glass funnel is fitted to the inflow pickup tubing, and an aspirator bulb is attached to the output manifold tubing for the purpose of drawing air samples through the system. As it is impracticable to attempt to pull the gas sample through all the test tubes at one time, pinchcocks are attached to each input tube as it comes off the intake manifold at the place marked by an X in figure 3. As each test is made, the pinchcock is released while the bulb is squeezed 20 to 30 times to pull the test sample of air through the test tube. The pinchcock is then closed, and the one on the next tube is opened while air is drawn through the reagent mixture in the bottom of that tube; this procedure is repeated until all the tests have been run. Most of the high-level confirmatory tests described below can be completed in this apparatus. Reagents for the identification of phosphates and fluorides in nerve gas should go in the first two test tubes. When the test for lewisite is run, a section of glass tubing containing acetylene indicator paper must be placed between the plastic stopper and the outflow manifold assembly. Reagents for the detection of mustard and phosgene should follow in that order. Additional high-level confirmatory tests can be run in this analyzer by simply adding more test tubes, each with the appropriate solution for that particular test. A cyanogen chloride test would be the most likely one to add next, since this war gas may give a positive reaction with some of the nerve gas test reagents, thereby causing confusion.

The Bournique apparatus (fig. 4) is used when liquid test reagents are required. Only one test can be run at a time. The reagent is placed in the Pyrex flask, which can be heated if necessary. The test vessel is then closed with the aspiratory assembly, and air is passed through the liquid in the vessel by

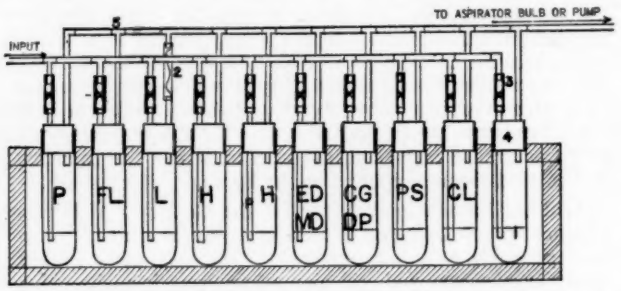

Fig. 3.—Aspirator unit of Lever air-flow apparatus. 1, 1 in. by 4 in. test tubes. 2, cuprous paper indicator. 3, Saran tubing connectors. 4, plastic stoppers, no. 5. 5, 5 mm. L and T glass tubing. X, pinch clamps. The tubes are labeled according to the recommended sequence of tests. This apparatus may be adapted in size to field use by eliminating some of the tests.

pressing and releasing the aspirator bulb 30 to 60 times. As this is done, the entrance tube of the aspiratory assembly or the optional collecting funnel with its impermeable plastic tubing connection is held near suspicious-looking droplets or surfaces. When contamination is present the ground, bushes, and other surfaces will often have a dark, spattered appearance.

The Bournique equipment shown in figure 2 again operates as an aspiration system, but the air to be tested is now pulled through small glass reaction tubes. The tubes may contain a solid that supports the reagent used, a solid that absorbs the war agent, or a test paper impregnated with the reagent. The reaction tubes are prepared by cutting 4 mm. glass tubing into 2 to 3 in. (5.1 to 7.6 cm.) lengths and fire-polishing at each end. A small amount of glass wool is inserted at one end and followed by the reagent, supported on a suitable solid in certain cases, by a solid adsorbent (such as silica gel) in other cases, or by strips of reagent-impregnated paper in still other instances. The exact nature of the filling used is indicated for each test below. At the other end of the reaction tube a second wad of glass wool may be inserted, if necessary. The tests are carried out by pulling air through each tube in the manner described for the apparatus in figure 4. In either case, the Bournique apparatus will give a change of color without further use of chemicals in an F test. In an H test, more reagents must be added or additional chemical operations carried out. Since the operator must be adequately protected with a gas mask and

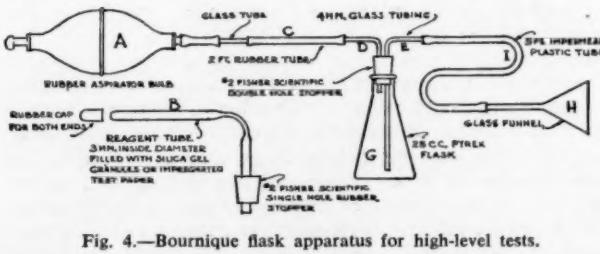

Fig. 4.—Bournique flask apparatus for high-level tests.

protective clothing, this will be difficult unless he retires to an upwind area that is free of contamination once the samples have been secured. The mask and some of the protective clothing may then be removed to facilitate additional operations. It is, therefore, wise, when preparing to do H testing, to approach an area of suspected contamination from upwind and establish a base outside of the contaminated area. Some of the bulkier equipment may be left at this point while the samples are gathered in the area of suspected contamination.

In an apparatus designed by Mr. Lever for expelling poisonous chemical agents from samples of material collected in the

field and collecting these agents for high-level laboratory identification, contaminated samples are placed at the bottom of a 4 oz. (0.12 liter) bottle with a two-hole rubber stopper. By means of glass tubing connection, air is blown down through one stopper vent over or into the specimen with an aspirator bulb; the air flows through a connecting tube that extends into the absorbent material for the chemical agent, which is contained at the bottom of a test tube fitted with another two-hole stopper, one hole accommodating the connecting tube and the other, an exhaust vent. Suitable high-level identification tests can then be carried out on the gas specimen concentrated in this absorbent material.

Morris B. Jacobs, Ph.D., Chief, Division of Chemical Detection, Department of Health, City of New York, has designed a chemical agents detector kit for use by the civil defense chemical officers in that city. Equipment and the detection procedures are included for the detection of mustard, phosgene, halogens, and nerve gases. The Jacobs kit may be purchased from the LaMar Laboratories, Inc., New York 10.

The various tests for the detection of chemical warfare agents will be described in the order in which testing should be carried out, the procedures for the most toxic and dangerous of the agents being given first. This order is: nerve gases such as fluorophosphates, vesicants or blister gases, choking or irritant gases, and blood gases. Details of preparation of the reagents to be used for the various tests follow the list of tests.

Detection of Fluorophosphates (Nerve Gases): Chemical warfare agents that inhibit the action of acetylcholinesterase in the body and act as nerve poisons will generally contain either fluorine or phosphorus, if not both. These agents may be detected by decomposition to either fluoride or phosphate ion and identification of these ions with suitable tests. Decomposition will generally be effected by heating with 5% sodium hydroxide solution, which results in hydrolysis to fluoride and phosphate. The tests involving such decomposition are of type H, owing to the further operations of heating and addition of reagents that are necessary.

1. Fluoride Ion Detection (H Test): Using the Bournique apparatus (fig. 4), 10 ml. of 5% sodium hydroxide solution is placed in the Pyrex flask. The contaminated material is either added to the flask or sampled with the aspiratory assembly as described above. The aspiratory assembly is then removed, and the flask contents are heated with a Bunsen burner or alcohol lamp for 5 to 10 minutes. After cooling, the solution is acidified with 2N hydrochloric acid (about 6.3 ml.) and is divided into two equal portions, which are placed in separate Pyrex flasks. A blank solution is prepared by adding 5 ml. of the sodium hydroxide solution to another Pyrex flask. The sodium hydroxide solution is acidified with about 3.3 ml. of 2N hydrochloric acid, and 2 ml. of zirconium nitrate-sodium alizarinate test solution is added to it. Two milliliters of zirconium nitrate-sodium alizarinate test solution is added to one of the two equal portions of the sample solution, and this is compared to the blank solution. If the sample solution shows any appreciable bleaching of color of the reagent as compared to the red color of the blank, fluoride is present.

2. Phosphate Ion Detection (H Test): To the second portion of the sample solution, 2 ml. of ammonium molybdate solution is added. The Pyrex flask is shaken and allowed to stand for a few moments; 1 ml. of hydroquinone reagent is added and the vessel shaken again; and finally, 1 ml. of 20% sodium sulfite solution is added and the flask shaken once more. If phosphate is present, its phosphomolybdate complex will be reduced to molybdenum blue, producing an intense blue color.

The tests for fluoride and phosphate ion may also be carried out with the Lever reaction tubes (fig. 3) or the Jacobs kit. The fluoride test in the Jacobs kit is carried out by use of a reaction tube containing a strip of paper that has been impregnated with zirconium nitrate and sodium alizarinate solutions. The paper is wet with 5% sodium hydroxide solution and the usual aspiration carried out to collect a sample. The reaction tube is then heated with an alcohol lamp in such a manner as not to char the paper. Fading of the paper from pink or red to white indicates fluoride. For the phosphate test, a plain strip of filter paper is placed in the reaction tube and wet with 5% sodium hydroxide solution. The sample is taken and the wet portion of the paper heated with the lamp as before. After cooling, 2 drops of 2N hydrochloric acid are added, followed by 1 drop of molybdate solution and 2 drops of ammonia benzidine as the reducing agent. The formation of a blue color indicates phosphate. To avoid trouble due to charring of paper in both tests, washed asbestos fiber might be used as absorbent in place of filter paper.

3. Detection of Blister Gases or Vesicants: Field detection is generally feasible for agents of this class, when present in liquid form, with a dye that dissolves in the liquid agent to give a distinctively colored solution. An example is Sudan red combinations, which give a red color when sprinkled on a liquid vesicant agent. MK.1 detector powder turns reddish or purple when dissolved in a vesicant. Since some organic solvents, sulfuric acid, etc. also dissolve such dyes to produce colors, such detection is subject to many false results and must be confirmed by H tests. It is necessary to make at least two such tests, one for the arsenic-containing vesicants and the other for the persistent mustard and nitrogen mustard chemical agents.

4. Differentiation Between Mustard and Nitrogen Mustard (F test): The only test now available other than the chloramide-Congo red test, is one based on a change in pH. The nitrogen mustards give an alkaline reaction. As a result, air containing such materials will give a blue color indicative of a pH of 9 to 11 when aspirated over moistened universal indicator paper in glass aspiration tubes, using the Bournique reaction tube apparatus. Persistent liquid vesicants could be directly tested with dry universal indicator paper. The color obtained in a positive test for nitrogen mustards will be blue. Other vesicant gases will give an acid reaction (red color) when passed over the moistened universal indicator paper, and other persistent vesicants in liquid form will react with the dry universal indicator paper to give a red color.

5. Detection of Mustard and Nitrogen Mustard with Selenium Dioxide (H Tests): Either the Lever air-flow or the Bournique flask apparatus may be used. Two milliliters of the selenium dioxide reagent is added to the Pyrex flask, the vessel tightly attached to the aspiration assembly, and a sample taken in the usual manner. The mixture in the vessel is then warmed nearly to boiling with an alcohol lamp or Bunsen burner. If mustard gases have been collected, the selenium dioxide will be reduced to free selenium with production of a red color; however, other reducing agents, such as hydrogen sulfide and, probably, the arsenical vesicants will produce the same results. This test may also be carried out with the Bournique reaction-tube apparatus. A Pyrex flask containing a mixture of calcium chloride powder and selenium dioxide is used for the aspiration technique by which the sample is gathered. Two drops of 2N hydrochloric acid are then added to the calcium chloride-selenium dioxide mixture. At this point it is advisable to heat the flask gently with the alcohol lamp or Bunsen burner, although some procedures do not give such instructions. Again, the production of a red color indicates the presence of mustard or nitrogen mustard.

6. Detection of Mustard and Nitrogen Mustard with Sodium Iodoplatinate: Using the Bournique reaction-tube apparatus, 1 drop of 5% acetic acid or of 2N hydrochloric acid is added to a reaction tube containing either silica gel or glass wool. Aspiration in the usual manner is done to obtain a sample; 3 drops of sodium iodoplatinate are added to the tube, followed by 1 drop of starch solution. Mustard and nitrogen mustard will reduce the iodoplatinate with liberation of free iodine, which reacts with the starch solution to give the characteristic blue starch-iodide complex. Chlorine, nitrous fumes, and arsenical vesicants may interfere.

7. Detection of Arsenical Vesicants (H Test): Five grams of arsenic-free zinc is placed in the Bournique flask and sufficient copper sulfate solution added to cover it. After a minute the copper solution is drained off and replaced with 10 ml. of water. Contaminated material is added to the flask or sampled by aspiration in the usual manner. A wad of cotton moistened with lead acetate solution is placed next to the constriction of the stoppered reagent tube; then a strip of mercuric bromide or mercuric chloride paper is inserted in the tube, with a little of the paper extending beyond the distal end of the tube to act as a comparison blank. The aspiration assembly is removed

from the Pyrex flask, 5 ml. of concentrated hydrochloric acid added to it, and the reagent tube tightly inserted into its neck. If arsenicals are present, arsine (AsH_3) will be evolved. This substance reacts with the mercuric salt of the test paper to produce yellowish-brown to black-brown complexes. The lead acetate solution on the cotton plug preceding the test paper is for the purpose of removing hydrogen sulfide, which would react with the test paper to form black lead sulfide. If the identification of individual arsenicals is desired, further H tests must be carried out.

Differentiation of Arsenical Vesicants: The following tests are all of type H but are also very simple and could probably be used virtually as F procedures.

8. Detection of Diphenylaminechlorarsine: Silica gel in one of the Bournique reaction tubes is impregnated with 2 to 3 drops of concentrated sulfuric acid, or several milliliters of this acid is placed in the Pyrex flask of the Bournique flask apparatus. In either case, aspiration is done in the usual manner or the suspected liquid is added. This war agent produces a red color with the cold acid. On addition of 2 to 3 drops of 5% potassium nitrate solution the color changes to purple or blue, probably owing to an impurity, diphenylamine, which is always present in this war gas. The other arsenicals give no color with sulfuric acid; the test is confirmatory for this chlorarsine.

9. Detection of Lewisite: The suspected vapor is aspirated in the usual manner through a Bournique reaction tube that contains silica gel, or suspected liquid is sucked up; (1) 2 to 3 drops of Sloat's reagent, an alkaline solution of cuprous ion, are then added. If lewisite is present it will form acetylene, and the acetylene will produce a red color by the formation of cuprous acetylide. This test is quite specific. An F procedure may be done by the insertion of a strip of filter paper moistened with the reagent in the reaction tube, followed by immediate aspiration. A red color again indicates lewisite. (2) Alternatively, the reaction tube is opened and some of the vapor-impregnated silica gel granules are permitted to drop into 1 or 2 ml. of ammoniacal silver nitrate solution. If lewisite is present, the silver compound is reduced and the granules will turn black.

10. Detection of Ethyl or Methyl Dichlorarsine: The suspected vapor is introduced in the Bournique reaction-tube apparatus as before; 2 to 3 drops of 5% mercurous nitrate solution are then added. Formation of a gray color within a few seconds indicates one of the above dichlorarsines. Lewisite will give a gray color after about 12 hours.

11. Detection of Diphenylcyanoarsine: The procedure for identifying dichlorarsines is carried out. On addition of the mercurous nitrate, the silica gel should turn black immediately if diphenylcyanoarsine is present. Alternatively, several silica gel granules used in sampling, as above, are dropped into 1 or 2 ml. of 5% mercurous nitrate solution acidified with a few drops of 0.1N nitric acid. Rapid blackening in the cold solution indicates diphenylcyanoarsine, while the more gradual development of a gray color indicates either methyl or ethyl dichlorarsine. It may be difficult to distinguish among these three agents without testing for cyanide.

Detection of Lung Irritants: As no specific reagents for this class of war gases appear to be available, it is necessary to test separately for the individual lung irritants.

12. Detection of Chlorine with Aniline Hydrochloride (F Test): The Bournique tube contains aniline hydrochloride paper rolled into spirals. As suspected air is aspirated through the tube in the usual manner, a color change from white to red or blue is observed in the presence of chlorine. However, many other oxidizing agents may interfere.

13. Detection of Phosgene, Di-Phosgene, and Tri-Phosgene (F Test): The Bournique reaction tube contains a strip of filter paper that has been impregnated with 2 or 3 drops of Harrison's reagent. On aspiration in the usual manner, the paper changes from colorless to yellow in the presence of phosgene. The following substances may interfere: hydrogen chloride, bromine, chlorine, lewisite, and nitrogen dioxide. To differentiate among these gases, the same test is done substituting 2 drops of nitroso reagent for the Harrison's reagent. If phosgene alone is present a Michler's type ketone is formed that causes the paper to change in color from brown to green. The nitroso reagent is virtually specific for phosgene. A positive test with Harrison's reagent and a negative with the nitroso reagent indicates di-phosgene and tri-phosgene but no phosgene. The nitroso reagent is rather difficult to prepare and may decompose within two or three months of preparation.

14. Detection of Chlorpicrin (H Test): (1) Nitrite reaction: 2 or 3 drops of sodium ethylate solution are added to the silica gel in a Bournique reaction tube, and aspiration is done in the usual manner. Any chlorpicrin that passes through the tube will be decomposed by the sodium ethylate with the formation of nitrite. After aspiration, 2 or 3 drops of glacial acetic acid, 1 or 2 drops of sulfanilic acid, and 1 or 2 drops of alpha-naphthylamine are added. The nitrite will diazotize the sulfanilic acid, which then couples with the alpha-naphthylamine to form a rose or pink color when chlorpicrin is present. Chloroform interferes. This test may also be carried out with the Bournique flask apparatus. With 1 or 2 ml. of sodium ethylate in the Pyrex flask, aspiration is done and several drops of glacial acetic acid are added to the flask, followed by 1 ml. each of sulfanilic acid and alpha-naphthylamine. The color change is as indicated above when chlorpicrin is present. (2) Test with diphenylamine sulfonate: with 1 or 2 drops of concentrated sulfuric acid on the silica gel of a Bournique reaction tube, a sample is collected as usual; 1 drop of water is added to the tube and allowed to stand for one minute; 1 drop of barium diphenylamine sulfonate reagent is added. If chlorpicrin is present, it will hydrolyze to nitrous acid, which reacts with the diphenylamine reagent to give a blue color. Oxides of nitrogen, stannic chloride, and titanium tetrachloride interfere. This test may also be carried out with the Bournique flask apparatus. With 2 or 3 ml. of concentrated sulfuric acid in the Pyrex flask, aspiration is carried out in the usual manner; several drops of water are added, followed one minute later by 2 drops of barium diphenylamine sulfonate reagent. Chlorpicrin gives a blue color as before.

Detection of Systemic Poisons: The two most likely agents of this type appear to be hydrogen cyanide and arsine. Tests for these materials are given below.

15. Detection of Hydrogen Cyanide (F Test): The Bournique reaction tube contains a spiral of filter paper that has been dipped into an equal mixture of copper acetate solution and benzidine acetate solution. Aspiration is done; in the presence of hydrogen cyanide, a blue color is produced on the indicator paper.

16. Detection of Arsine (F Test): This test is the same as that used for arsenical vesicants (test 7) from the point at which the latter gases have been decomposed into arsine. The test is readily carried out with the Bournique reaction tube apparatus. The tube now contains mercuric chloride or bromide paper preceded by a cotton plug moistened with lead acetate solution. The suspected vapor first passes over the lead acetate, which will remove hydrogen sulfide when present in the air sample; any arsine that is present will react with the mercuric bromide or chloride to form a yellow or brown complex on the test paper.

General Tests for Chemical Agents: A number of general methods have been proposed for the detection of war agents. Some of these are quite elaborate and depend on combustion procedures that decompose the war gas into easily recognized products; others depend on simpler techniques. The two most readily applied simple procedures follow.

17. pH Test for War Gases (F Test): This test depends on the acid or alkaline reactions given by various chemical agents as they decompose by hydrolysis to form free acids and bases. In some cases it is only necessary to touch suspected liquid with a piece of universal pH paper. In other cases the aspiration of suspected vapor through a solution of universal pH indicator in a Lever test tube or a Bournique flask may be more effective. Substances that hydrolyze to give an acid reaction include mustard, phosgene, and cyanogen chloride. These will produce a red color with the indicator. As already mentioned, nitrogen mustards will give an alkaline reaction and have a blue color.

18. Beilstein Test for War Gases (F Test): A copper wire heated in a flame such as that of an alcohol lamp will give a green flame if a war gas containing halogen or cyanide is present in the atmosphere surrounding the lamp. The wire must be heated in halogen-free air until green color is absent from the flame before making the test.

Reagents.—Directions follow for the preparation of the various reagents that are used in the tests described above.

Reagents for the Fluoride Test (test 1): (1) Sodium hydroxide 5%: dissolve 5 gm. of solid sodium hydroxide in 100 ml. of water. (2) 2N hydrochloric acid: dilute the concentrated acid with 5 vol. of water to each volume of acid. (3) Zirconium nitrate-sodium alizarinate solution: dissolve 0.87 gm. of zirconium nitrate [$Zr(NO_3)_2.5H_2O$] in 100 ml. of water. Dissolve 0.17 gm. of sodium alizarinate in 100 ml. of water. Mix equal portions of the two solutions just prior to use. (4) Zirconium nitrate-sodium alizarinate test paper: the solutions of each of the reagents are prepared, as above, just before use. Place the zirconium nitrate solution in one evaporating dish and the sodium alizarinate solution in another. Circles of filter paper are first placed in the sodium alizarinate solution for a few seconds and then dipped into the zirconium nitrate solution. After drying, the paper is cut into strips 2 in. (5.08 cm.) long and about ¼ in. (0.64 cm.) wide. The strips are rolled into spirals, and one strip is inserted into each end of a reaction tube. At one end of the tube the paper is pushed back about ½ in. to permit the addition of liquid reagents.

Reagents for the Phosphate Test (test 2): (1) Ammonium molybdate solution (Bournique): dissolve 25 gm. of ammonium molybdate in 300 ml. of water. Dilute 75 ml. of concentrated sulfuric acid to 200 ml. with distilled water and add to the molybdate solution. (2) Ammonium molybdate solution (Jacobs): dissolve 40 gm. of ammonium molybdate in 100 ml. of water. (3) Hydroquinone solution: dissolve 0.5 gm. of hydroquinone in 100 ml. of water and add a drop of concentrated sulfuric acid to retard oxidation. (4) Ammonia benzidine solution: dissolve 0.05 gm. of benzidine or benzidine hydrochloride in 10 ml. of concentrated acetic acid and dilute with water to 100 ml. Add 7 ml. of concentrated ammonium hydroxide solution to 21 ml. of the benzidine solution.

Reagent for Nitrogen Mustard Test (test 4): Universal pH indicator solution or paper may be obtained from virtually any chemical supply house.

Reagents for Mustard Gas Test (test 5): (1) Selenium dioxide-calcium chloride reaction tubes: mix, preferably by thoroughly grinding in a mortar with a pestle, 5 gm. of selenium dioxide with 20 gm. of powdered anhydrous calcium chloride. Fill reaction tubes to a length of about 1 to 1½ in. (2.54 to 3.81 cm.) with this mixture, using glass wool to close each end. This is the mustard gas reagent used in the Jacobs kit. (2) 5% sodium nitrate solution: dissolve 5 gm. of the salt in 100 ml. of water.

Reagents for Mustard Gas Test (test 6): (1) Sodium iodoplatinate solution: add 1 ml. of a 5% platinic chloride solution to 5.3 ml. of a 5% solution of sodium iodide. Dilute to 180 ml. with water. (2) Starch solution: mix about 1 gm. of soluble starch with 4 to 6 ml. of water until a paste is formed. Add 200 ml. of boiling water and stir thoroughly. Cool and add 2 drops of toluene as a preservative. (3) 2N hydrochloric acid: see reagents for test 1. (4) 5% acetic acid: dilute 5 ml. of glacial acetic acid with 95 ml. of water.

Reagents for the Arsenical Test (test 7): (1) Concentrated hydrochloric acid. (2) Arsenic-free zinc shot. (3) Copper sulfate solution: dissolve 5 gm. of the pure hydrate in 100 ml. of water. (4) Lead acetate solution: dissolve 5 gm. of the pure salt in 100 ml. of water. (5) Mercuric chloride or mercuric bromide paper: dissolve 10 gm. of mercuric chloride or mercuric bromide in 100 ml. of water; immerse a circle of Whatman filter paper no. 40 in the solution, allow to dry, and cut into strips. The mercuric chloride and mercuric bromide paper may also be purchased from a supply house.

Reagents for Detection of Lewisite (test 9): (1) Sloat's reagent: dissolve 10 gm. of sodium hydroxide in 50 ml. of water. Add to this solution 4 gm. of arsenic trioxide and stir until it is in solution. Prepare a second solution of 0.4 gm. of copper acetate dihydrate [$Cu(C_2H_3O_2)_2H_2O$] in 10 ml. of water. Heat both solutions to 85 C, and add the copper solution to the arsenic trioxide solution with constant stirring. Cool the mixture rapidly in cold water; the orange precipitate should now dissolve, and the solution may be diluted with 40 ml. of water to a final volume of 100 ml. (2) Ammoniacal silver nitrate solution: dilute concentrated ammonium hydroxide 1:1 with water and add this, drop by drop, to 10 ml. of a silver nitrate solution containing 5 gm. of the salt per 100 ml. of water until the precipitate that first forms is completely dissolved.

Reagent for Detection of Ethyl or Methyl Dichlorarsine and Diphenylcyanoarsine (tests 10 and 11): 5% mercurous nitrate solution: this reagent should be freshly prepared by dissolving 5 gm. of the pure salt in 100 ml. of water.

Reagents for Phosgene Tests (test 13): (1) Harrison's reagent: dissolve 5 gm. of *p*-dimethylaminobenzaldehyde and 5 gm. of colorless diphenylamine each in 50 ml. of carbon tetrachloride. Combine the two solutions immediately and dip filter paper into the mixture. The filter paper is allowed to dry and is cut into thin strips, which are stored in a tightly-sealed, brown, dry bottle. For use, one or two strips may be twisted into a spiral and pushed into an empty reaction tube, or the strips may be directly exposed to the suspected air. (2) Nitroso reagent: it is first necessary that 1,2,4,nitrosodiethylaminophenol be prepared; 0.5 gm. of m-diethylaminophenol (obtainable from Eastman Kodak Co.) is dissolved in 15 ml. of water containing 1 ml. of concentrated hydrochloric acid. The solution must be cooled to 0 C before use. Sodium nitrite, 0.22 gm., is dissolved in 5 ml. of water and added to the first solution with an eyedropper while the temperature is constantly maintained at 0 C. The addition should require 20 minutes. The mixture is then made nearly neutral by the addition of a solution of 2 gm. of sodium acetate in 5 ml. of water, after which it is extracted with 50 ml. of benzene. After it separates, the benzene layer is filtered through a dry filter paper. The final reagent is prepared by mixing equal portions of the filtered benzene layer and a solution of 0.2 gm. of m-diethylaminophenol in 50 ml. of benzene. The reagent is kept tightly stoppered in a brown bottle. For use, 2 or 3 drops are added to a twisted strip of filter paper pushed into an empty reaction tube. The reagent is unstable and must be renewed about every three months.

Reagents for the Chlorpicrin Test (test 14): (1) Sodium ethylate solution: dissolve 3 gm. of sodium in 100 ml. of ethyl alcohol. (2) Sulfanilic acid solution: dissolve 1 gm. of sulfanilic acid in hot water, cool, and dilute to 100 ml. (3) Alpha-naphthylamine hydrochloride solution: boil 0.5 gm. of this substance in 100 ml. of water for 10 minutes. Add water to keep the volume essentially constant during the boiling period. (4) Barium diphenylamine sulfonate solution: dissolve 2 gm. of this material in 100 ml. of water. If on standing a blue color develops, a fresh solution must be prepared.

Reagents for the Hydrogen Cyanide Test (test 15): (1) Cupric acetate solution: dissolve 2.86 gm. of copper acetate dihydrate in water and dilute to 1 liter. (2) Benzidine acetate solution: place an excess of benzidine acetate in 475 ml. of water. Shake or stir until the solution is saturated. Filter and dilute to 1 liter with 525 ml. of distilled water. (3) Impregnated paper: strips of filter paper 3 to 4 mm. wide should be dipped in an equal mixture of the above two solutions just before sampling is done.

Reagents for the Arsine Test (test 16): See above, reagents for test 7.

COMMENT

The procedures described above will probably permit the satisfactory detection of any one of the war gases if only that agent is present. In the case of certain mixtures of gases, satisfactory results may still be obtained. With very complicated mixtures this may no longer be the case. It is not very likely that extremely complicated mixtures would be used, but the possibility cannot be discounted. Moreover, many other agents have been proposed as war gases concerning which little is known. Should some of these prove especially potent and be adapted to wartime use, it is probable that the tests that have been given would prove inadequate. As a result, the scheme proposed here can be regarded only as a tentative one that might be subject to radical revision in the event of actual hostilities. Civil defense might then depend on the release of adequate procedures for detection by the armed forces. Another alternative would be the study of this problem of a satisfactory scheme for detection of war gases by a suitable national organization. It is probably too large a problem to be adequately treated by any one state civil defense group.

MEDICAL NEWS

ARKANSAS

Society News.—Dr. Columbus R. Ellis, Malvern, recently became president of the Arkansas Academy of General Practice; Dr. Benjamin N. Saltzman, Mountain Home, was made president-elect; and Dr. Clifton C. Long, Ozark, was reelected secretary-treasurer.——Newly elected officers of the Arkansas Obstetrical and Gynecological Society include: Dr. Charles P. Wickard, Little Rock, president; Dr. John W. Jones, Texarkana, vice-president; and Dr. J. F. Kelsey, Fort Smith, secretary.

Personal.—Dr. Fount Richardson, 316 W. Dickson, Fayetteville, has been named editor of the *Journal of the Arkansas Medical Society,* succeeding Dr. William R. Brooksher, who resigned after 20 years of service.——Dr. William King Jordan, Little Rock, head of the department of neurology, University of Arkansas School of Medicine, took part in the International Neurochemical Symposium held in Oxford University, England, during July.

Rockefeller Rural Survey.—The Arkansas Medical Society has appointed the following physicians as an advisory committee to work with the University of Arkansas School of Medicine, Little Rock, on the Rockefeller plan for a study of Arkansas rural health facilities: chairman, Louis K. Hundley, Pine Bluff, who is chairman of the society's council; William R. Brooksher, Fort Smith, president of the society; James M. Kolb, Clarksville; Fount Richardson, Fayetteville; Benjamin N. Saltzman, Mountain Home; and Henry W. Thomas, Dermott. The study, to be financed by Mr. Winthrop Rockefeller, who proposed the plan, will include the establishment of a pilot clinic.

CALIFORNIA

Town Honors Physicians.—A new elementary school in Turlock is to bear the name of two of the town's family physicians, Drs. Albert E. and Eric A. Julien, aged 73 and 71 years, respectively, chosen because they have "given unstintingly of their time . . . to help others."

Public Relations Director.—In a move to strengthen its public relations program, the Los Angeles County Medical Association has appointed Jerry Pettis, former associate director of public relations for the California Medical Association, as executive assistant to the president. Mr. Pettis, who served as assistant to the president of United Air Lines before joining the state medical organization, is the founder of Audio-Digest (THE JOURNAL, March 20, 1954, page 1007; June 2, 1954, page 73), a nonprofit C. M. A. subsidiary that he serves as executive vice-president.

Postgraduate Surgical Clinics.—Starting Sept. 14, 8 to 10 p. m., and continuing for six weeks at weekly intervals, postgraduate surgical clinics will be given at Fort Miley by the department of surgery of the Veterans Administration Hospital and Stanford University School of Medicine, San Francisco. Members of the American Academy of General Practice will receive 12 hours' education credit. Nonmembers are invited to attend on payment of the registration fee. Interns and resident staffs of the Bay Area hospitals are welcome to attend without fee but must register. Dr. Forrest M. Willett, chief of medical service at Fort Miley and associate professor of medicine at Stanford, will be coordinator of the program, and Dr. Louis G. Brizzolara, chief of surgical service at Fort Miley and assistant clinical professor of surgery at the University of California School of Medicine, San Francisco, will act as moderator. Those wishing to obtain program and registration forms should contact Dr. Alexander F. Fraser, program chairman, 3004 16th St., San Francisco, HEmlock 1-0388, or the office of the California Academy of General Practice, 461 Market St., San Francisco, YUkin 2-2329.

Physicians are invited to send to this department items of news of general interest, for example, those relating to society activities, new hospitals, education, and public health. Programs should be received at least three weeks before the date of meeting.

GEORGIA

Dr. Russell Oppenheimer Retires.—Dr. Russell H. Oppenheimer, who for the past 33 years has been associated with the Emory University School of Medicine, Emory University, retired as professor of medicine and director of postgraduate education at Emory Aug. 31. He has been dean of the medical school for 20 years and superintendent of the university hospital for 13 years. He served with the Army in World War I and, after a period of private practice in Detroit, joined the staff of Grady Memorial hospital in Atlanta as the first resident physician in the Emory University division in 1921. Dr. Oppenheimer has been a leader in medical-civic affairs. When Battle Hill Haven, a home for the aged and chronically ill, opened in Atlanta, he volunteered to supervise the medical care of patients without compensation. He has also served on the board of trustees of the Atlanta Tuberculosis Association and on the medical committee of the Social Planning Council. Dr. Oppenheimer is an honorary member of Phi Rho Sigma and Alpha Omega Alpha societies. He plans to make his home with his sister, Mrs. Eleanor M. Bronson, in Jacksonville, Fla.

IDAHO

State Medical Election.—Newly elected officers of the Idaho State Medical Association include Drs. Alexander Barclay Jr., Coeur d'Alene, president; Robert S. McKean, Boise, president-elect; and Quentin W. Mack, Boise, secretary-treasurer. The next annual meeting will be held at Sun Valley, June 19 to 22, 1955.

ILLINOIS

Society News.—Newly elected officers of the Illinois Psychiatric Society include Dr. Percival Bailey, Chicago, president; Dr. Franz G. Alexander, Chicago, vice-president; and Dr. Alex J. Arieff, Chicago, secretary-treasurer.

Chicago

Cancer Fellowship Established.—The Bertha Gerber Memorial Cancer League, of which Dr. Adolph A. Smolen is president, has established the Bertha Gerber Memorial Cancer League fellowship at the Chicago Medical School with a grant of $3,000 for the present year. The money is to be used to support a young scientist engaged in cancer research in the department of oncology under Philippe Shubik, Ph.D., director.

Institute on Rheumatic Fever.—La Rabida Sanitarium, Chicago, announces an institute on rheumatic fever and rheumatic heart disease, Oct. 11 to 15, directed primarily to the general practitioner or family physician and to nurses, medical social workers, occupational therapists, dentists, and others with a similar interest. The last day will be devoted to a scientific program, concluding with the Robert A. Black lecture on rheumatic fever. Advance registration will be required for those who wish to attend the entire five day session. Visitors to individual sessions will be admitted by card on previous application. Information will be supplied by circular on application to Institute, La Rabida Sanitarium, E. 65th St. and South Shore Dr., Chicago 49.

MARYLAND

Alvarenga Prize Awarded.—The College of Physicians of Philadelphia on July 14 awarded the Alvarenga prize for 1954 to Dr. DeWitt Stetten Jr., associate director in charge of research, National Institute of Arthritis and Metabolic Diseases, Bethesda, for contributions to the knowledge of metabolic diseases. The Alvarenga prize was established by the will of Pedro Francisco DaCosta Alvarenga of Lisbon, Portugal, an associate fellow of the College of Physicians of Philadelphia, to be awarded annually by the College of Physicians on the anniversary of the death of the testator, July 14, 1883.

Personal.—An honorary doctor of science degree has been conferred on Charles W. Shilling, Captain, M.C., U. S. N., senior medical officer of the U. S. Naval Academy, Annapolis, by Taylor University of Upland, Ind., "because of his scientific contributions to the field of military medicine." Captain Shilling's primary interest has been in the field of submarine medicine and deep sea diving. In 1939 his knowledge was put to use as the senior medical officer in charge of the rescue of the crew of the stricken submarine U.S.S. Squalus.——Dr. Charles R. Hayman, Bel Air, who has been serving as deputy state and county health officer since 1949, has gone to Juneau, Alaska, to assume his duties as deputy commissioner of health and chief of the medical services section, Alaska Department of Health.

MICHIGAN

Dr. Kennedy Honored.—A testimonial dinner in honor of Dr. Robert B. Kennedy, associate professor of obstetrics, Wayne University College of Medicine, Detroit, held in Detroit, June 30, was attended by over 225 friends, fellow physicians, and their wives and was sponsored by Dr. Kennedy's former residents, who trained under him while he was chief of the department of obstetrics at Woman's Hospital and St. Joseph Mercy Hospital in Detroit. Dr. Carl F. Shelton was chairman of the dinner. Drs. William E. Johnston of Woman's Hospital and John P. Hubbard Jr. of St. Joseph Mercy Hospital were the toastmasters. Dr. Charles S. Stevenson represented Wayne University. A scroll from the mayor of Detroit and the key to the city were presented to Dr. and Mrs. Kennedy.

MINNESOTA

Society News.—The Minnesota Academy of Occupational Medicine and Surgery, which was organized Oct. 1, 1953, held its first annual meeting May 26 at the Minneapolis Athletic Club. Dr. John F. Shronts, Minneapolis, was named president; Dr. Leslie W. Foker, Minneapolis, vice-president; Dr. James R. Fox, Minneapolis, secretary; Dr. Tracy E. Barber, Austin, treasurer; and Dr. Gordon C. MacRae, Duluth, recorder.

Pediatric Reunion to Honor Dr. McQuarrie.—A pediatrics reunion at the University of Minnesota, Minneapolis, will be held in conjunction with the annual Northwestern Pediatric Society meeting, Sept. 23 to 25, in honor of Dr. Irvine McQuarrie, head, department of pediatrics, who has given 25 years of service to the university. Lectures will be delivered at the meeting by 22 academic leaders in the field of pediatrics, most of whom received their pediatric training under Dr. McQuarrie. As an additional tribute, a McQuarrie Pediatrics Fund has been established with a goal of $50,000. The annual income will be used to provide a lectureship in pediatrics, a scientific travel fund for pediatrics staff members to attend special conferences, fellowships in unusual circumstances, and equipment that may be needed from time to time by the department, and for any other purpose within the department as approved by the committee. Before coming to Minnesota, Dr. McQuarrie served on staffs of the University of California Medical School, Berkeley-San Francisco, Henry Ford Hospital, Detroit, Yale University, New Haven, Conn., and the University of Rochester (N. Y.) School of Medicine and Dentistry. Dr. McQuarrie is editor in chief of Brennemann's "Practice of Pediatrics."

NEBRASKA

Narcotic Violation.—Dr. Charles Wells Neill, South Sioux City, was found guilty of having violated federal narcotic laws and on June 30 was sentenced to serve a term of one year and one day.

Crippled Children's Clinics.—The following clinics have been scheduled for crippled children:

Sept. 11, Broken Bow, High School.
Sept. 25, Lexington, High School.
Oct. 9, O'Neill, High School.
Oct. 23, Wayne, Benthack Hospital.

NEW JERSEY

Society News.—Dr. Richard B. Cattell, director, Lahey Clinic, Boston, will be the guest speaker at the installation dinner of the Passaic County Medical Society, Sept. 15, at the Alexander Hamilton Hotel in Passaic. His subject will be "Surgical Diseases of the Pancreas."——At the recent annual meeting of the Radiological Society of New Jersey the following officers were elected: Dr. Nicholas G. Demy, Plainfield, president; Dr. Solomon Silvera, Jersey City, vice-president; Dr. Carye-Belle Henle, Newark, secretary; and Dr. Leonard S. Ellenbogen, Atlantic City, treasurer.

NEW MEXICO

State Medical Election.—At the annual meeting of the New Mexico Medical Society the following officers were elected: Drs. John F. Conway, Clovis, president; Stuart W. Adler, Albuquerque, president-elect; Earl L. Malone, Roswell, vice-president; and Lewis M. Overton, Albuquerque, secretary-treasurer.

NEW YORK

"Operation Ragweed."—The major phase of "Operation Ragweed" was recently completed in Sullivan County in a county-wide drive jointly sponsored by the board of supervisors and the Sullivan County Hotel Association. Specially equipped motor trucks sprayed 3,560 miles of state, county, and town roadside with 100,000 gallons of pollen-killing chemicals. Spray equipment was fitted with floodlights to facilitate night operation during the week-long effort.

University News.—State University of New York College of Medicine at Syracuse announces the following promotions: from assistant professor to associate professor of pathology, Drs. John T. Prior and Martin F. Hilfinger Jr.; from clinical assistant professor to clinical associate professor of medicine, Dr. William G. Woodin; and from instructor to assistant professor, Dr. Barbara R. Rennick (physiology), Dr. Francis S. Caliva (medicine), Dr. William H. Bergstrom (pediatrics), and Dr. Harold B. Houser (medicine).——Dr. Leonard D. Fenninger, formerly chief of the section of general medicine of the National Cancer Institute, Bethesda, Md., has been appointed assistant dean and assistant professor of medicine at his alma mater, the University of Rochester School of Medicine and Dentistry, where from 1947 to 1952 he was a fellow in cancer research and an instructor in medicine. During 1952 and 1953, Dr. Fenninger was a U. S. Public Health Service surgeon.

Society News.—The New York Rheumatism Association recently elected Dr. Robert L. Preston, president; Dr. Mortimer E. Ehrlich, vice-president; and Dr. Bernard Rogoff, secretary-treasurer (reelected).

Foreign Guests on Cardiology Program.—The scientific session of the New York Heart Association, Inc., Sept. 21, 8:30 p. m., will be held in Hosack Hall, New York Academy of Medicine Building, 2 E. 103rd St. "Total and Local Blood Volumes in Congestive Heart Failure" will be presented by Dr. Gustave Nylin, Stockholm, Sweden, and "Surgery of Acquired and Congenital Heart Disease" by Dr. Russell C. Brock, London, England.

New York City

Courses on Physical Therapy.—The Institute of Physical Medicine and Rehabilitation, New York University-Bellevue Medical Center, and the New York University School of Education are again offering (Nov. 22 to Dec. 17; Feb. 7 to March 4, 1955; May 2 to May 27) a four week advanced course in physical rehabilitation methods with four points of university credit. Tuition fee is $80; registration fee, $8. Qualified veterans may enroll in the course under the provision of P. L. 346 or P. L. 16. The course, which will cover (1) severe disabilities and their rehabilitation, (2) skills and methods of functional activities, and (3) clinical experience, is open to certified physical therapists who have the approval of the curriculum director. Applications and requests for information may be submitted to Mrs. Edith

Buchwald Lawton, Director of Rehabilitation Courses for Physical Therapists, Institute of Physical Medicine and Rehabilitation, 400 E. 34th St., New York 16.

NORTH CAROLINA

Personal.—Dr. Francis Bayard Carter, professor of obstetrics and gynecology, Duke University School of Medicine, Durham, has been named an honorary fellow of the Society of Obstetricians and Gynecologists of Canada. Dr. Carter is president of the American Academy of Obstetrics and Gynecology and past president of the South Atlantic Obstetrical and Gynecological Society. At Duke's 102nd commencement in June, he became the third faculty recipient of the Algernon Sydney Sullivan award.——The President's Committee on Employment of the Physically Handicapped has bestowed awards on Dr. Oscar Lee Miller, Charlotte, and Dr. William M. Roberts, Gastonia.

Physicians Address Foreign Societies.—Dr. R. Wayne Rundles and Dr. Samuel P. Martin III, associate professors of medicine at Duke Hospital and Duke University School of Medicine, Durham, will address the International Congress of Hematology at Paris, France, Sept. 10. Dr. Martin will discuss "Leukocyte Metabolism" and Dr. Rundles, "Abnormal Proteins in Malignant Blood Disease." Dr. Martin, one of Duke's seven Markle scholars, also will address a symposium in London, Oct. 8, sponsored by the Ciba Foundation, discussing "Metabolic Response to Infection." He will visit medical schools in Belgium, France, Switzerland, Germany, Denmark, Sweden, and England before returning to Duke, Oct. 18. Dr. Rundles will visit the Royal Cancer Society and other blood laboratories in London and Paris as well as protein laboratories in Sweden before his return late in September. A member of the Duke staff since 1945, Dr. Rundles is the author of numerous articles on neuroanatomy, diabetes, and blood diseases.

SOUTH CAROLINA

Personal.—For outstanding service to the community of Greenwood, Dr. Clough H. Blake has been awarded the Rotary Club's "Man of the Year" plaque.——Dr. Rudolph Farmer, assistant superintendent and medical director of the South Carolina Sanatorium, State Park, has been appointed superintendent to succeed Col. William H. Moncrief, who recently resigned.

Pediatricians Meet in Columbia.—The South Carolina Pediatric Society will meet Sept. 13 and 14 in Columbia. At the Monday meeting, a joint session with the Columbia Medical Society at 7 p. m., Dr. Clifford G. Grulee Jr., associate professor of pediatrics, Tulane University of Louisiana School of Medicine, New Orleans, will present "The Diagnosis and Treatment of Diphtheria—A Continuing Pediatric Problem" and Dr. Harry B. O'Rear, professor of pediatrics, University of Georgia School of Medicine, Augusta, "Treatment of Meningococcic Infections in Children." The Tuesday session will begin at 11 a. m. Dr. Grulee will discuss "Infectious Hepatitis in Children," and Dr. O'Rear will have as his topic "Cat Scratch Disease."

Dr. Peeples Named State Health Officer.—Dr. George S. T. Peeples, Columbia, assistant health officer and director of local health services for the state board of health, was recently appointed state health officer to succeed the late Dr. Benjamin F. Wyman. Dr. Peeples, a county health officer in Horry and Dillon counties in 1926 and 1927, becoming assistant director of local health services in 1935, was appointed director of the division of crippled children in 1942 and in 1949 was named local health services director. He received the degree of master of public health from Harvard University in 1937. During World War II he was commissioned a surgeon by the U. S. Public Health Service. He is a former president of the State Public Health Association. Dr. Clarence L. Guyton, Columbia, has been appointed assistant health officer.

TENNESSEE

Society News.—Newly elected officers of the Tennessee Radiological Society are: Dr. J. Marsh Frere, Chattanooga, president; Dr. John M. Wilson, Memphis, vice-president; and Dr. George K. Henshall, Chattanooga, secretary-treasurer.

Dr. Raines Named Department Head.—Dr. Samuel L. Raines, assistant professor in the department of urological surgery at his alma mater, the University of Tennessee College of Medicine, Memphis, has been advanced to professor and named head of the department. Dr. Raines has been a member of the college of medicine staff since 1929. He succeeds Dr. Thomas D. Moore, who has resigned as head of the department.

TEXAS

Course in Electrocardiology.—A course in practical electrocardiology will be presented, Dec. 6 to 10, at the University of Texas Postgraduate School of Medicine, Houston, with the cosponsorship of Baylor University College of Medicine and the Houston Heart Association. Dr. Demetrio Sodi-Pallares, chief of the department of electrocardiology, National Institute of Cardiology of Mexico City, will be guest lecturer each evening from 7:30 to 10 p. m. During the daytime hours, registrants will be invited to attend the various hospitals in the Texas Medical Center during periods when routine hospital tracings are being read by the hospital electrocardiologists. In addition, registrants in smaller groups will be given individualized instruction in interpretation of specific tracings by various members of the faculties of both schools. The course, which includes adult and pediatric electrocardiology, is primarily designed to acquaint general practitioners as well as internists with practical aspects of electrocardiology, with emphasis on those tracings commonly encountered in practice. Further inquiry and advance registration may be made through the University of Texas Postgraduate School of Medicine (telephone: LI-6986).

The New John Sealy Hospital.—This hospital, recently dedicated at the University of Texas Medical Branch, Galveston, together with the R. Waverley Smith Memorial Pavilion, covers some-

John Sealy Hospital

what over 10 acres. The entire building, with the exception of the pavilion, is dedicated to the functions of a teaching hospital. The operating department, which serves the entire hospital, is designed in a "radial" pattern. A circumferential corridor circling this operating complex provides a "front" entrance to the operating, scrub-up, and anesthesia rooms as well as the spectators' galleries, of which every major room has one. Among other special facilities are a fever therapy suite of two treatment and two rest rooms; separate suites for electrocardiography and basal metabolism, each accommodating three patients simultaneously; a blood bank with a five place donors' room; and a processing laboratory. The total construction cost of the hospital exceeded 11 million dollars, about 9 million of which was contributed by the Sealy and Smith Foundation for the John Sealy Hospital.

WASHINGTON

Annual Alfred Strauss Lecture.—The department of surgery, University of Washington School of Medicine, Seattle, will again sponsor the annual Alfred A. Strauss lecture, which will be presented by Dr. Lester R. Dragstedt, chairman, department of surgery, University of Chicago School of Medicine, on Oct. 22 at 8:15 p. m. in the auditorium of the Health Sciences Building, University of Washington campus. Dr. Dragstedt will discuss "Etiology of Gastric and Duodenal Ulcer." Dr. Dragstedt was previously affiliated with the physiology and pharmacology

departments of the State University of Iowa and served as Nathan Smith Davis professor of physiology and pharmacology and head of the department at Northwestern University Medical School. He received the A. M. A. silver medal in 1947 for his work on vagotomy in the treatment of peptic ulcer and the A. M. A. gold medal in 1950 for his exhibit on quantitative studies on mechanisms of gastric secretion. Physicians, medical students, nurses, and others working with the medical profession are welcome to attend the Strauss lecture.

WEST VIRGINIA

Hospital News.—Morris Memorial Hospital for Crippled Children at Milton has received an endowment of $10,000 in U. S. treasury bonds. The gift was made by Arthur B. Koontz, Charleston, on behalf of the estate of his late brother, Patrick B. Koontz.

Society News.—Dr. Edith L. Potter, associate professor of pathology, University of Chicago School of Medicine, and pathologist to the Chicago Lying-In Hospital, will be the guest speaker at the meeting of Kanawha Medical Society, Sept. 14, in the evening. The speaker will discuss "Perinatal Morbidity and Foetal Salvage" and will lead a round-table discussion at Memorial Hospital in Charleston at 4 p. m. Members of all other component societies in the state are invited to this opening meeting of the fall season.

Rural Health Conference.—The seventh annual Rural Health Conference, sponsored by the West Virginia State Medical Association, was held in the Century of Progress Hall at Jackson's Mill, Sept. 2, under the auspices of the public relations committee, with the cooperation of the West Virginia Farm Bureau, the agricultural extension division of West Virginia University, and the state department of health. Dr. Charles E. Staats, Charleston, chairman, public relations committee, presided as moderator. After an address of welcome by Dr. Russel Kessel, Charleston, president of the state medical association, Mrs. Dean Johnson, Buckhannon, president of the Farm Women's Council, presented "Health Problems of Rural People," and Mr. Edgar F. Fisher Jr., Richmond, director, Virginia Council on Health and Medical Care, discussed the part rural groups have played in assuring the success of the program of his organization in Virginia. At the morning session Dr. Carll S. Mundy, Toledo, Ohio, vice-chairman of the A. M. A. Council on Rural Health, presented "Roads to Rural Health." The afternoon meeting was devoted to discussion by separate groups on nutrition, medical and hospital service plans, nursing problems, prevention and control of disease, and medical care in rural areas. Physicians who served as moderators included Dr. Newman H. Dyer, Charleston, state director of health, and Dr. William L. Cooke, Charleston, past president, Kanawha Medical Society. An invitation to attend the meeting was extended to members of farm groups. The West Virginia State Medical Association was host at a luncheon in the Mt. Vernon dining hall.

WISCONSIN

Bronze Bust of Dr. Schweitzer.—A bronze bust of Dr. Albert Schweitzer, the gift of a group of hospital staff members and other Wisconsin residents to the University of Wisconsin, was recently unveiled in the lobby of the University Hospitals, Madison. The plaque on the bust reads: "Versatile scholar in theology and philosophy. Distinguished contemporary interpreter of Bach. Founder and for 40 years missionary surgeon of the hospital at Lamberence, French Equatorial Africa. By his surpassing achievements in the service of God and his fellowmen, Dr. Schweitzer has pointed the way to a full life in medicine. In his example the weak will find strength, and the stalwart renewed courage and resolution." Among those participating in the unveiling were Dr. William S. Middleton, dean of the medical school; University of Wisconsin president Edwin B. Fred; and Dr. Noel A. Gillespie, associate in anesthesiology at University Hospitals, who, 30 years ago, worked with Dr. Schweitzer at Lamberene. In accepting the bust, President Fred said Dr. Schweitzer "has carved from the jungle a remarkable oasis for the Africans among whom he has lived for the past 41 years. He has saved thousands of lives with none of the appurtenances modern doctors find indispensable. . . . It is especially fitting that it be placed here, where lives are saved every day by dedicated men, where young men are trained by precept and example to dedicate themselves to the saving of lives in the future."

GENERAL

Meeting on Pharmacology and Experimental Therapeutics.—The interim meeting of the American Society for Pharmacology and Experimental Therapeutics will be held at Charlottesville, Va., Sept. 6 to 8, under the joint sponsorship of the University of Virginia School of Medicine, Charlottesville, and the Medical College of Virginia, Richmond. On Monday at 1:30 p. m. there will be a panel discussion on graduate education in pharmacology and on Tuesday at 9:15 a. m., a panel discussion on the cellular localization of drugs and drug effects. In all, 151 additional presentations have been scheduled, and 16 papers will be read by title. The pharmacology dinner will be held Tuesday, 7 p. m., in the gymnasium.

Fellowships for Research in Arthritis.—The Arthritis and Rheumatism Foundation is offering the following research fellowships in the basic sciences related to arthritis:

1. Predoctoral fellowships ranging from $1,500 to $3,000 per annum, depending on the family responsibilities of the fellow, tenable for one year with prospect of renewal.
2. Postdoctoral fellowships ranging from $4,000 to $6,000 per annum, depending on family responsibilities, tenable for one year with prospect of renewal.
3. Senior fellowships for more experienced investigators, carrying an award of $6,000 to $7,500 per annum, tenable for five years.

The deadline for applications is Oct. 15. Applications will be reviewed and awards made in January, 1955. Information may be had from the Medical Director, The Arthritis and Rheumatism Foundation, 23 W. 45th St., New York 36.

Interim American Board of Occupational Medicine.—This board is negotiating with the American Board of Preventive Medicine relative to the grouping of occupational medicine with other nationally recognized professional specialties in the general field of preventive medicine. For purposes of organization, it is desirable to know the number of candidates likely to apply for certification in occupational medicine under such auspices. Interested physicians are invited to communicate with Dr. Carl M. Peterson, Secretary, Interim American Board of Occupational Medicine, 535 N. Dearborn St., Chicago 10. Application forms and other essential information will be sent to inquirers as negotiations proceed. Full instructions will include regulations governing basic eligibility, admission to certification without examination, examination procedure, fees, and other details.

Cerebral Palsy Institutes.—The national office of United Cerebral Palsy and its affiliates throughout the country have scheduled eight program institutes in September and October. Each institute will start on Thursday noon and extend through Friday evening, immediately preceding regional conventions in each area. The institutes and conventions have been scheduled as follows:

Sept. 2-5, Pacific Region, St. Francis Hotel, San Francisco.
Sept. 9-12, Midwest Region, Hotel Muehlebach, Kansas City, Mo.
Sept. 16-19, Central Region, Hotel Cleveland, Cleveland.
Sept. 23-26, New England Region, Statler Hotel, Boston.
Sept. 30-Oct. 3, Eastern Region, Benjamin Franklin Hotel, Philadelphia.
Oct. 7-10, Southeast Region, Heidelberg Hotel, Jackson, Miss.
Oct. 14-17, Southwest Region, Roosevelt Hotel, New Orleans.
Oct. 21-24, Southern Region, John Marshall Hotel, Richmond, Va.

A summary of the program institute discussions will be presented on Saturday afternoon. On Saturday evening the guest speaker will discuss "Legislation for the Physically Handicapped." The U. C. P. 1954 annual convention is scheduled to be held at the Mayflower Hotel in Washington, D. C., Nov. 19 to 21.

Meeting of Physiologists.—The sixth annual fall meeting of the American Physiological Society will be held at the University of Wisconsin, Madison, Sept. 8 to 10, when 272 papers will be presented. Session chairmen will include Dr. Herbert S. Gasser, until recently director of the Rockefeller Institute for Medical Research, New York, and Dr. Carl J. Wiggers, editor of *Circulation Research*, formerly head of the department of physi-

ology at Western Reserve University School of Medicine, Cleveland. On Sept. 6 and 7, (1) a refresher course in the physiology of respiration will be conducted by Dr. Julius H. Comroe Jr., professor of physiology and pharmacology at the University of Pennsylvania Graduate School of Medicine, Philadelphia, and Hermann Rahn, Ph.D., associate professor of physiology at the University of Rochester (N. Y.) School of Medicine and Dentistry; (2) a review of a long-range survey of the physiological sciences, sponsored by the American Physiological Society, will reveal the results of a two year comprehensive study of physiology as a profession; and (3) members of the Physiology Study Section, U. S. Public Health Service, will meet to consider applications for grants-in-aid for research projects.

Prevalence of Poliomyelitis.—According to the National Office of Vital Statistics, the following number of reported cases of poliomyelitis occurred in the United States and its territories and possessions in the weeks ended as indicated:

Area	Aug. 7, 1954: Paralytic Type	Aug. 7, 1954: Total Cases Reported	Aug. 8, 1953, Total
New England States			
Maine	2	5	8
New Hampshire	..	..	8
Vermont	..	..	8
Massachusetts	4	14	19
Rhode Island	..	4	10
Connecticut	1	13	24
Middle Atlantic States			
New York	13	50	125
New Jersey	22	36	51
Pennsylvania	..	40	53
East North Central States			
Ohio	30	104	119
Indiana	18	39	46
Illinois	41	78	117
Michigan	30	95	158
Wisconsin	2	19	30
West North Central States			
Minnesota	8	28	139
Iowa	25	84	48
Missouri	16	30	58
North Dakota	1	5	14
South Dakota	..	1	5
Nebraska	22	32	12
Kansas	4	29	18
South Atlantic States			
Delaware	3	4	1
Maryland	5	8	32
District of Columbia	2	3	7
Virginia	22	39	47
West Virginia	6	10	39
North Carolina	15	54	51
South Carolina	4	15	10
Georgia	16	41	26
Florida	16	60	22
East South Central States			
Kentucky	15	48	28
Tennessee	5	54	62
Alabama	13	19	29
Mississippi	8	29	22
West South Central States			
Arkansas	4	9	21
Louisiana	10	18	18
Oklahoma	11	34	36
Texas	53	152	81
Mountain States			
Montana	1	4	6
Idaho	..	4	4
Wyoming	2	11	7
Colorado	6	16	16
New Mexico	2	11	7
Arizona	..	7	31
Utah	..	8	8
Nevada	..	9	5
Pacific States			
Washington	5	14	14
Oregon	4	9	13
California	118	211	157
Territories and Possessions			
Alaska	8	18	..
Hawaii	1	3	3
Puerto Rico	..	..	..
Total	594	1,633	1,873

Society News.—Newly elected officers of the **American Dermatological Association** include Dr. Richard S. Weiss, St. Louis, president; Dr. Clark W. Finnerud, Chicago, vice-president; and Dr. J. Lamar Callaway, Durham, N. C., secretary.——At the annual meeting of the **Society for Investigative Dermatology,** Dr. Clarence S. Livingood, Detroit, was elected president and Dr. J. Walter Wilson, Los Angeles, vice-president.——At its annual meeting the **American Proctologic Society** elected the following officers: Dr. A. W. Martin Marino, Brooklyn, N. Y., president; Dr. Stuart T. Ross, Hempstead, N. Y., president-elect; Dr. Kenneth E. Smiley, Los Angeles, vice-president; Dr. Rufus C. Alley, Lexington, Ky., treasurer; and Dr. Karl Zimmerman, Pittsburgh, secretary.——At the annual business meeting of the **American Psychosomatic Society** the following physicians took office: Dr. Lawrence S. Kubie, New York, president; Dr. Stanley Cobb, Boston, president-elect; and Dr. Theodore Lidz, New Haven, Conn., secretary-treasurer. The next annual meeting will be held May 4 and 5, 1955, at the Claridge Hotel in Atlantic City, N. J.——Officers of the **American Radium Society** for 1954-1955 include: Dr. John E. Wirth, Pasadena, Calif., president; Dr. Grant H. Beckstrand, Long Beach, Calif., president-elect; Dr. Norman A. McCormick, Windsor, Ontario, Canada, first vice-president; Dr. W. Edward Chamberlain, Philadelphia, second vice-president; Dr. Robert E. Fricke, Rochester, Minn., secretary; and Dr. Douglas J. Roberts, Hartford, Conn., treasurer.——The annual meeting of the **North Pacific Society of Neurology and Psychiatry** was held at the Harrison Hot Springs Hotel, Harrison Hot Springs, British Columbia, Canada, April 2 and 3. Dr. Harold G. Wolff, New York, guest speaker, presented "Headache Mechanisms" and "Participation of the Nervous System in Processes Altering Vulnerability of Tissue." The following officers were elected for 1954: Dr. William Y. Baker, Seattle, president; Dr. Wendell H. Hutchens, Portland, Ore., president-elect; Dr. John W. Evans, Portland, Ore., secretary-treasurer; and Drs. Robert S. Dow, Portland, Ore., Douglas E. Alcorn, Victoria, B. C., and Donald E. Stafford, Seattle, executive committee.

LATIN AMERICA

New Medical Journal.—*Revista da associação medica brasileira,* the official organ of the Brazilian medical profession, published its first issue in March. The 128 page journal contains original articles, clinical notes, editorials, correspondence, news, and abstracts. The postal address of this quarterly publication is Caixa Postal 8.904, São Paulo, S. P., Brazil.

CORRECTION

Mosher Life-Saver.—In a footnote in THE JOURNAL, Aug. 7, 1954, page 1297, the proper name Mosher was misspelled. The pharyngeal airway referred to was named after Dr. Harris P. Mosher, professor emeritus at Harvard Medical School.

MEETINGS

AMERICAN MEDICAL ASSOCIATION: Dr. George F. Lull, 535 North Dearborn St., Chicago 10, Secretary.
1954 Clinical Meeting, Miami, Florida, Nov. 29-Dec. 2.
1955 Annual Meeting, Atlantic City, N. J., June 6-10.
1955 Clinical Meeting, Boston, Nov. 29-Dec. 2.
1956 Annual Meeting, Chicago, June 11-15.
1956 Clinical Meeting, Seattle, Nov. 27-30.

ACADEMY OF PSYCHOSOMATIC MEDICINE, New York, Oct. 8-9. Dr. Ethan Allan Brown, 75 Bay State Road, Boston 15, Secretary.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, The Waldorf-Astoria, New York, Sept. 19-24. Dr. W. L. Benedict, 100 First Avenue Bldg., Rochester, Minn., Executive Secretary.

AMERICAN ACADEMY OF PEDIATRICS, Palmer House, Chicago, Oct. 4-7. Dr. E. H. Christopherson, 610 Church St., Evanston, Ill., Secretary.

AMERICAN ASSOCIATION OF BLOOD BANKS, The Shoreham, Washington, D. C., Sept. 13-16. Miss Marjorie Saunders, 3500 Gaston Ave., Dallas 4, Texas, Secretary.

AMERICAN ASSOCIATION OF MEDICAL RECORD LIBRARIANS, Sheraton-Cadillac Hotel, Detroit, Oct. 4-8. Miss Doris Gleason, 510 N. Dearborn St., Chicago 10, Executive Director.

AMERICAN ASSOCIATION OF OBSTETRICIANS, GYNECOLOGISTS AND ABDOMINAL SURGEONS, The Homestead, Hot Springs, Va., Sept. 9-11. Dr. Frank R. Lock, Bowman Gray School of Medicine, Winston-Salem, N. C., Secretary.

AMERICAN CANCER SOCIETY, Hotel Roosevelt, New York, Oct. 17-24. Dr. Charles S. Cameron, 47 Beaver St., New York 4, Medical Director.

AMERICAN CLINICAL AND CLIMATOLOGICAL ASSOCIATION, Lake Placid Club, Lake Placid, N. Y., Oct. 14-16. Dr. Marshall N. Fulton, 124 Waterman St., Providence 6, R. I., Secretary.

AMERICAN COLLEGE OF GASTROENTEROLOGY, The Shoreham, Washington, D. C., Oct. 25-30. Dr. A. Xerxes Rossien, 33 West 60th St., New York 23, Secretary.

AMERICAN CONGRESS OF PHYSICAL MEDICINE AND REHABILITATION, Hotel Statler, Washington, D. C., Sept. 6-11. Dr. Walter J. Zeiter, 30 N. Michigan Ave., Chicago 2, Executive Director.

AMERICAN FRACTURE ASSOCIATION, Shamrock Hotel, Houston, Texas, Oct. 11-14. Dr. H. W. Wellmerling, 626 Griesheim Bldg., Bloomington, Ill., Secretary-General.

AMERICAN HEART ASSOCIATION, Jung Hotel, New Orleans, Oct. 26-30. Mr. Irving Hexter, 44 East 23d St., New York 10, Secretary.

AMERICAN HOSPITAL ASSOCIATION, Palmer House, Chicago, Sept. 13-16. Dr. E. L. Crosby, 18 East Division St., Chicago 10, Director.

AMERICAN MEDICAL WRITERS' ASSOCIATION, Hotel Sherman, Chicago, Sept. 24. Dr. Harold Swanberg, 510 Maine St., Quincy, Ill., Secretary.

AMERICAN OTORHINOLOGIC SOCIETY FOR PLASTIC SURGERY, The Waldorf-Astoria, New York, Sept. 19. Dr. Louis J. Feit, 66 Park Ave., New York, Secretary.

AMERICAN PUBLIC HEALTH ASSOCIATION, Memorial Auditorium, Buffalo, N. Y., Oct. 11-15. Dr. Reginald M. Atwater, 1790 Broadway, New York 19, Executive Secretary.

AMERICAN ROENTGEN RAY SOCIETY, Shoreham Hotel, Washington, D. C., Sept. 21-24. Dr. Barton R. Young, Germantown Hospital, Philadelphia 44, Secretary.

AMERICAN SOCIETY OF ANESTHESIOLOGISTS, Netherland-Plaza Hotel, Cincinnati, Oct. 25-30. Dr. J. Earl Remlinger Jr., 188 West Randolph St., Chicago 1, Secretary.

AMERICAN SOCIETY OF CLINICAL PATHOLOGISTS, Shoreham Hotel, Washington, D. C., Sept. 6. Dr. Clyde G. Culbertson, 1040-1232 W. Michigan St., Indianapolis, Secretary.

ASSOCIATION OF LIFE INSURANCE MEDICAL DIRECTORS OF AMERICA, Royal York Hotel, Toronto, Canada, Oct. 12-14. Dr. Henry B. Kirkland, P. O. Box 594, Newark, N. J., Secretary.

ASSOCIATION OF AMERICAN MEDICAL COLLEGES, French Lick Springs Hotel, French Lick, Ind., Oct. 17-20. Dr. Dean F. Smiley, 185 N. Wabash Ave., Chicago 1, Secretary.

CALIFORNIA ACADEMY OF GENERAL PRACTICE, Statler Hotel, Los Angeles, Oct. 24-27. Mr. Wm. W. Rogers, 450 Mission St., San Francisco, Executive Secretary.

CENTRAL ASSOCIATION OF OBSTETRICIANS AND GYNECOLOGISTS, Hotel Jefferson, St. Louis, Oct. 7-9. Dr. Harold L. Gainey, Suite 602, 116 S. Michigan Ave., Chicago 3, Secretary.

CENTRAL SOCIETY FOR CLINICAL RESEARCH, Drake Hotel, Chicago, Oct. 29-30. Dr. Robert H. Ebert, 950 East 59th St., Chicago 37, Secretary.

CLINICAL ORTHOPAEDIC SOCIETY, Sheraton Hotel, Chicago, Oct. 7-9. Dr. John H. Moe, 825 Nicollet Ave., Minneapolis, Secretary.

COLLEGE OF AMERICAN PATHOLOGISTS, The Shoreham, Washington, D. C., Sept. 6. Dr. Arthur H. Dearing, 203 N. Wabash Ave., Chicago 1, Executive Secretary.

COLORADO STATE MEDICAL SOCIETY, Broadmoor Hotel, Colorado Springs, Sept. 21-24. Mr. Harvey T. Sethman, 835 Republic Building, Denver 2, Executive Secretary.

DELAWARE, MEDICAL SOCIETY OF, Dover, Oct. 11-13. Dr. Norman L. Cannon, 1208 Delaware Ave., Wilmington, Executive Secretary.

GULF COAST CLINICAL SOCIETY, Edgewater Park, Miss., Oct. 21-22. Dr. F. C. Minkler, Pascagoula, Miss., Secretary.

INDIANA STATE MEDICAL ASSOCIATION, Murat Temple, Indianapolis, Oct. 26-28. Mr. James A. Waggener, 23 East Ohio St., Indianpolis 4, Executive Secretary.

INDUSTRIAL HEALTH CONFERENCE (Houston), Shamrock Hotel, Houston, Tex., Sept. 23-25. Dr. Sidney Schnur, 411 Medical Arts Bldg., Houston 2, Tex., Chairman.

KANSAS CITY SOUTHWEST CLINICAL SOCIETY, Kansas City, Mo., Oct. 4-7. Dr. Ira C. Layton, 306 E. Twelfth St., Kansas City 6E, Mo., Secretary.

KENTUCKY STATE MEDICAL ASSOCIATION, Brown Hotel, Louisville, Sept. 21-23. Dr. Bruce Underwood, 620 S. Third St., Louisville 2, Secretary.

MICHIGAN STATE MEDICAL SOCIETY, Sheraton-Cadillac Hotel, Detroit, Sept. 29-Oct. 1. Dr. L. Fernald Foster, 606 Townsend St., Lansing 15, Secretary.

MIDWESTERN SECTION OF AMERICAN FEDERATION FOR CLINICAL RESEARCH, Thorne Hall Auditorium, Northwestern University Medical Campus, Chicago, Oct. 28. Dr. R. L. Grissom, Univ. of Nebraska College of Medicine, Dept. of Internal Medicine, Omaha 5, Secretary.

MISSISSIPPI VALLEY MEDICAL SOCIETY, Hotel Sherman, Chicago, Sept. 22-24. Dr. Harold Swanberg, 510 Maine St., Quincy, Ill., Secretary.

MONTANA MEDICAL ASSOCIATION, Hotel Finlen, Butte, Sept. 16-19. Mr. L. R. Hegland, 1236 N. 28th St., Billings, Executive Secretary.

NATIONAL ASSOCIATION FOR MENTAL HEALTH, Hotel Statler, New York, Oct. 23-25. Mr. Robert M. Heininger, 1790 Broadway, New York 19, Executive Director.

NATIONAL PROCTOLOGIC ASSOCIATION, Maryland Hotel, Chicago, Oct. 7-9. Dr. George E. Mueller, 59 E. Madison St., Chicago 2, Executive Secretary.

NEW HAMPSHIRE MEDICAL SOCIETY, Mt. Washington Hotel, Bretton Woods, Oct. 3-5. Dr. W. H. Butterfield, 18 School St., Concord, Secretary.

NORTHERN MINNESOTA MEDICAL ASSOCIATION, Willmar, Sept. 10-11. Dr. C. L. Oppegaard, Crookston, Secretary.

OMAHA MID-WEST CLINICAL SOCIETY, Paxton Hotel, Omaha, Oct. 25-28. Dr. Louis E. Moon, 1031 Medical Arts Bldg., Omaha 2, Secretary.

OREGON STATE MEDICAL SOCIETY, Heathman Hotel, Portland, Oct. 13-16. Dr. Charles E. Littlehales, 1115 S. W. Taylor St., Portland 5, Executive Secretary.

PENNSYLVANIA, MEDICAL SOCIETY OF THE STATE OF, Bellevue-Stratford Hotel, Philadelphia, Oct. 17-22. Dr. Harold B. Gardner, 230 State St., Harrisburg, Secretary.

REGIONAL MEETINGS, AMERICAN COLLEGE OF PHYSICIANS:

Bismarck, N. D., Sept. 11. Dr. Robert B. Radl, 221 Fifth St., Bismarck, N. D., Governor.

Midwest, Indianapolis, Claypool Hotel, Oct. 9. Dr. Wendell A. Shullenberger, 3470 Central Ave., Indianapolis, Chairman.

New England, Hartford, Conn., Oct. 22. Dr. John C. Leonard, 80 Seymour St., Hartford, Conn., Chairman.

Southeastern, Edgewater Gulf Hotel, Edgewater Park, Miss., Oct. 15-16. Dr. E. Dice Lineberry, 1529 N. 25th St., Birmingham 4, Ala., Governor.

SOUTHWESTERN SURGICAL CONGRESS, Skirvin Hotel, Oklahoma City, Sept. 20-22. Dr. C. R. Rountree, 1227 Classen Drive, Oklahoma City 3, Secretary.

THE CONSTANTINIAN SOCIETY, The Broadmoor, Colorado Springs, Colo., Sept. 26-29. Dr. C. F. Shook, P. O. Box 1035-36, Toledo 1, Ohio, Secretary.

U. S. CHAPTER, INTERNATIONAL COLLEGE OF SURGEONS, Chicago, Sept. 7-10. Dr. Karl Meyer, 1516 Lake Shore Dr., Chicago, Secretary.

VERMONT STATE MEDICAL SOCIETY, Mt. Washington Hotel, Bretton Woods, N. H., Oct. 3-5. Dr. James P. Hammond, 337 South St., Bennington, Secretary.

WASHINGTON STATE MEDICAL ASSOCIATION, Davenport Hotel, Spokane, Sept. 18-22. Dr. Bruce Zimmerman, 1309 Seventh Ave., Seattle 1, Secretary.

WESTERN ASSOCIATION OF RAILWAY SURGEONS, Sun Valley, Idaho, Sept. 23-25. Dr. Leo L. Stanley, 1322 Fifth Ave., San Rafael, Calif., Secretary.

WISCONSIN, STATE MEDICAL SOCIETY OF, Hotel Schroeder, Milwaukee, Oct. 5-7. Mr. Charles H. Crownhart, 704 E. Gorham St., Madison 3, Secretary.

FOREIGN AND INTERNATIONAL

COMMONWEALTH HEALTH AND TUBERCULOSIS CONFERENCE, Royal Festival Hall, London, England, June 21-25, 1955. Mr. J. H. Harley Williams, Tavistock House North, Tavistock Square, London, W.C.1, England, Secretary General.

CONFERENCE OF INTERNATIONAL UNION AGAINST TUBERCULOSIS, Madrid, Spain, Sept. 26-Oct. 2, 1954. Secretariat, Escuela de Tisiologia, Ciudad Universitaria, Madrid, Spain.

CONFERENCE OF THE SOLVAY INSTITUTE OF SOCIOLOGY, Universiti Libre de Bruxelles, Brussels, Belgium, Oct. 18-23, 1954. For information write: Assistant to the Secretary, A. Dorsinfang Smets, Solvay Institute of Sociology, Parc Leopold, Brussels 4, Belgium.

CONGRESS OF INTERNATIONAL ASSOCIATION OF APPLIED PSYCHOLOGY, London, England, July 18-23, 1955. Dr. C. B. Frisby, National Institute of Industrial Psychology, 14 Welbeck St., London, W.1, England, President.

CONGRESS OF INTERNATIONAL ASSOCIATION FOR THE PREVENTION OF BLINDNESS, New York, N. Y., U. S. A., Sept. 12-17, 1954. Professor Franceschetti, 2 avenue Mirmot, Geneva, Switzerland, Secretary-General.

CONGRESS OF THE INTERNATIONAL DIABETES FEDERATION, Cambridge, England, July 4-8, 1955. Mr. James G. L. Jackson, 152 Harley St., London, W.1, England, Executive Secretary General.

CONGRESS OF INTERNATIONAL SOCIETY OF MEDICAL HYDROLOGY, Vichy, Paris, and Enghien, France, Sept. 24-27, 1954. Dr. Francon, 55, rue des Mathurins, Paris 8°, France, Secretary-General.

HEALTH CONGRESS OF THE ROYAL SANITARY INSTITUTE, Bournemouth, England, April 26-29, 1955. Mr. P. Arthur Wells, Royal Sanitary Institute, 90 Buckingham Palace Road, London, S.W.1, England, Secretary.

INTER-AMERICAN CONGRESS OF RADIOLOGY, Shoreham Hotel, Washington, D. C., U. S. A., April 24-29, 1955. Dr. Eugene P. Pendergrass, 3400 Spruce St., Philadelphia 4, Pa., U. S. A., Secretary-General.

INTER-AMERICAN SESSION, AMERICAN COLLEGE OF SURGEONS, Universidad Mayor de San Marcos de Lima, Lima, Peru, S. A., Jan. 11-14, 1955. Dr. Michael L. Mason, 40 East Erie St., Chicago 11, Ill., U. S. A., Secretary.

INTERNATIONAL ANATOMICAL CONGRESS, Paris, France, July 25-30, 1955. Prof. Gaston Cordier, 45, rue des Saints-Pères, Paris 6°, France, Secretary-General.

INTERNATIONAL ANESTHESIA RESEARCH SOCIETY, Ambassador Hotel, Los Angeles, Calif., U. S. A., Oct. 10-14, 1954. For information write: Dr. T. H. Seldon, 102-110 Second Avenue S.W., Rochester, Minn., U. S. A.

INTERNATIONAL CONGRESS OF BIOCHEMISTRY, Brussels, Belgium, Aug. 1-6, 1955. Prof. C. Liebecq, 17 Place Delcour, Liege, Belgium, Secretary-General.

INTERNATIONAL CONGRESS OF CLINICAL PATHOLOGY, Washington, D. C., U. S. A., Sept. 6-10, 1954. Dr. Robert A. Moore, University of Pittsburgh, Schools of the Health Profession, Pittsburgh 13, Pa., U. S. A., Chairman, Committee on Arrangements.

INTERNATIONAL CONGRESS OF COMPARATIVE PATHOLOGY, Lausanne, Switzerland, May 26-31, 1955. Professor Hauduroy, 19 rue Cesar Roux, Lausanne, Switzerland, Secretary-General.

INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST, Barcelona, Spain, Oct. 4-8, 1954. Mr. Murray Kornfeld, 112 East Chestnut St., Chicago 11, Ill., U. S. A., Executive Secretary.

INTERNATIONAL CONGRESS OF HEMATOLOGY, Paris, Sept. 6-11, 1954. Dr. Jean Bernard, 86 rue d'Assas, Paris 6e, France, Secretary.

INTERNATIONAL CONGRESS OF THE HISTORY OF MEDICINE, Rome and Salerno, Italy, Sept. 13-20, 1954. For information write: Segreteria XIV Congresso Internazionale di Storia della Medicina, Instituto di Storia della Medicine, Citta Universitaria, Rome, Italy.

INTERNATIONAL CONGRESS OF HYDATID DISEASE, Madrid, Spain, Sept. 25-30, 1954. Dr. Jesus Calvo Melendro, Hospital Provincial, Sorea, Spain, Secretary-General.

INTERNATIONAL CONGRESS OF INDUSTRIAL MEDICINE, Naples, Italy, Sept. 13-19, 1954. Professor Scipione Caccuri, Director, Institute of Industrial Medicine Policlinico, Naples, Italy, Chairman, Organizing Committee.

INTERNATIONAL CONGRESS OF INTERNAL MEDICINE, Stockholm, Sweden, Sept. 15-18, 1954. Professor Anders Kristenson, Karolinska Sjukhuset, Stockholm 60, Sweden, Secretary-General.

INTERNATIONAL CONGRESS OF MILITARY MEDICINE AND PHARMACY, Luxemburg, Luxemburg, Nov. 7-12, 1954. Colonel A. R. Vernengo, Direcion General de Sanidad Militar, Pozos 2045, Buenos Aires, Argentina, S. A., Secretary-General.

INTERNATIONAL CONGRESS OF NUTRITION, Amsterdam, Netherlands, Sept. 13-17, 1954. Dr. M. van Eekelen, Centraal Instituut voor Voedingsonderzoek T.N.O., 61 Catharynesingel, Utrecht, Netherlands, General Secretary.

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY, University of Montreal and McGill University, Montreal, Canada, Sept. 9-11, 1954, and Waldorf-Astoria, New York, N. Y., U. S. A., Sept. 12-17, 1954. Dr. William L. Benedict, 100 First Avenue Building, Rochester, Minn., U. S. A., Secretary-General.

INTERNATIONAL CONGRESS OF PLASTIC SURGERY, Stockholm, Sweden, Aug. 1-4, 1955, and Uppsala, Sweden, Aug. 5, 1955. Dr. Tord Skoog, Uppsala, Sweden, General Secretary.

INTERNATIONAL FEDERATION OF MEDICAL STUDENT ASSOCIATIONS, Rome, Italy, Oct. 1-5, 1954. Mr. Jorgen Falck Larsen, 12, Kristianiagade, Copenhagen Ø, Denmark, General Secretary.

INTERNATIONAL HOSPITAL CONGRESS, Lucerne, Switzerland, May 30-June 3, 1955. Capt. J. E. Stone, International Hospital Federation, 10 Old Jewry, London, E.C.2, England, Hon. Secretary.

INTERNATIONAL POLIOMYELITIS CONGRESS, University of Rome, Orthopedic Clinic, Rome, Italy, Sept. 6-10, 1954. Mr. Stanley E. Henwood, 120 Broadway, New York 5, N. Y., U. S. A., Executive Secretary.

INTERNATIONAL SOCIETY OF BLOOD TRANSFUSION, Paris, France, Sept. 12-19, 1954. For information write: Colonel Julliard, Société Internationale de Transfusion Sanguine, 53 boulevard Diderot, Paris 12e, France.

INTERNATIONAL SOCIETY FOR CELL BIOLOGY, Leiden, Netherlands, Sept. 1-8, 1954. Professor Peter J. Gaillard, University of Leiden, Leiden, Netherlands, Secretary.

INTERNATIONAL SOCIETY OF GEOGRAPHICAL PATHOLOGY, Washington, D. C., U. S. A., Sept. 6-10, 1954. Professor Fred C. Roulet, Hebelstrasse 24, Basel, Switzerland, Secretary-General.

INTERNATIONAL SURGICAL CONGRESS, Geneva, Switzerland, May 23-26, 1955. Dr. Max Thorek, 1516 Lake Shore Drive, Chicago, Illinois, U. S. A., Secretary-General.

JAPAN MEDICAL CONGRESS, Kyoto University and Kyoto Prefectural Medical College, Kyoto, Japan, April 1-5, 1955. Dr. Mitsuharu Goto, University Hospital, Medical Faculty of Kyoto University, Kyoto, Japan, Secretary-General.

LATIN AMERICAN CONGRESS OF ANESTHESIOLOGY, Sao Paulo, Brazil, S. A., Sept. 12-18, 1954. Dr. Zairo E. G. Vieira, Praca Floriano, 55-7°, And., Rio de Janeiro, Brazil, S. A., Secretario.

LATIN AMERICAN CONGRESS OF PHYSICAL MEDICINE, Lima, Peru, S. A., Feb. 14-19, 1955. Dr. Cassius Lopez de Victoria, 176 East 71st St., New York 21, N. Y., U. S. A., Executive Director.

MEDICAL JOURNALISM MEETING, Exposition Universelle Romaine, Rome, Italy, Sept. 30, 1954. Dr. H. Clegg, B.M.A. House, Tavistock Square, London W.C.1, England, Secretary.

MEDICAL WOMEN'S INTERNATIONAL ASSOCIATION CONGRESS, Lake Garda, Italy, Sept. 15-21, 1954. Dr. Ada Chree Reid, 118 Riverside Drive, New York 24, N Y., U. S. A., President.

PAN-AMERICAN ACADEMY OF GENERAL PRACTICE, Lima, Peru, S. A., Feb. 11-25, 1955. Dr. Arturo Martinez, 54 East 72d St., New York 21, N. Y. U. S. A., Secretary.

PAN AMERICAN HOMEOPATHIC MEDICAL CONGRESS, Hotel Gloria, Rio de Janeiro, Brazil, S. A., Oct. 2-13, 1954. Dr. Paul S. Schantz, 103 West Main St., Ephrata, Pa., U. S. A., Executive Secretary.

PAN-PACIFIC SURGICAL CONGRESS, Honolulu, Hawaii, Oct. 7-18, 1954. Dr. F. J. Pinkerton, Suite 7, Young Bldg., Honolulu 13, Hawaii, Director General.

WORLD CONGRESS OF CARDIOLOGY, Washington, D. C., U. S. A., Sept. 12-18, 1954. Dr. L. W. Gorham, 44 East 23d St., New York 10, N. Y., U. S. A., Secretary-General.

WORLD CONGRESS OF INTERNATIONAL SOCIETY FOR THE WELFARE OF CRIPPLES, Scheveningen-The Hague, Netherlands, Sept. 13-17, 1954. Secretariat: Miss H. P. Post, Pieter Lastmarkade 37, Amsterdam Z, Netherlands.

WORLD MEDICAL ASSOCIATION, Rome, Italy, Sept. 26-Oct. 2, 1954. Dr. Louis H. Bauer, 345 East 46th St., New York 17, N. Y., U. S. A., Secretary-General.

EXAMINATIONS AND LICENSURE

NATIONAL BOARD OF MEDICAL EXAMINERS: *Parts I and II.* Held in approved medical schools where there are five or more candidates. Dates: Sept. 7-8 (Part I only). Candidates may file examinations at any time but the National Board must receive them at least six weeks before the date of the examination they wish to take. New candidates should apply by formal registration; registered candidates should notify the board by letter. Sec., Dr. John P. Hubbard, 133 S. 36th St., Philadelphia 4.

BOARDS OF MEDICAL EXAMINERS

ARIZONA:* *Examination and Reciprocity.* Phoenix, Oct. 13-15, Jan. 12-14, 1955 and April 13-15, 1955. Ex. Sec., Mr. Robert Carpenter, 401 Security Bldg., Phoenix.

CALIFORNIA: *Written.* San Diego, Aug. 23-26; Sacramento, Oct. 18-21. *Oral.* San Diego, Aug. 21; Los Angeles, Nov. 20. *Oral and Clinical Examination for Foreign Medical School Graduates.* Sec., Dr. Louis E. Jones, 1020 N Street, Sacramento.

ILLINOIS: *Examination and Reciprocity.* Chicago, Oct. 5-7. Supt. of Registration, Mr. Frederic B. Selcke, Capitol Bldg., Springfield.

MINNESOTA:* *Examination and Reciprocity.* Minneapolis, Oct. 19-21. Sec., Dr. E. M. Jones, 230 Lowry Medical Arts Bldg., St. Paul 2.

MONTANA: *Examination and Reciprocity.* Helena, Oct. 4. Sec., Dr. S. A. Cooney, 214 Power Block, Helena.

NEW HAMPSHIRE: *Examination and Reciprocity.* Concord, Sept. 8-9. Sec., Dr. John S. Wheeler, 107 State House, Concord.

NEW MEXICO:* *Examination and Reciprocity.* Santa Fe, Oct. 11-12. Sec., Dr. R. C. Derbyshire, 227 E. Palace Ave., Santa Fe.

NORTH CAROLINA: *Endorsement.* Rocky Mount, Oct. 4. Sec., Dr. Joseph J. Combs, Professional Building, Raleigh.

OHIO: *Examination.* Columbus, Dec. 13-15. *Reciprocity.* Columbus, Oct. 4. Sec., Dr. H. M. Platter, Wyandotte Bldg., Columbus 15.

OREGON:* *Examination and Reciprocity.* Portland, Oct. 14-16. Exec. Sec., Mr. Howard I. Bobbitt, 609 Failing Building, Portland 4.

TENNESSEE:* *Examination.* Memphis, Sept. 29-30. Sec., Dr. H. W. Qualls, 1635 Exchange Bldg., Memphis.

TEXAS:* *Examination and Reciprocity.* Fort Worth, Dec. 2-4. Sec., Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth 2.

WEST VIRGINIA: *Examination.* Charleston, Oct. 11. Sec., Dr. Newman H. Dyer, State Office Bldg. No. 3, Charleston 5.

WISCONSIN:* *Reciprocity.* Madison, Oct. 15. Sec., Dr. Thomas W. Tormey, Room 1140, State Office Bldg., Madison 2.

WYOMING: *Examination and Reciprocity.* Cheyenne, Oct. 4. Sec., Dr. Franklin D. Yoder, State Office Bldg., Cheyenne.

ALASKA:* On application. Sec., Dr. W. M. Whitehead, 172 South Franklin St., Juneau.

GUAM: The Commission on Licensure will meet whenever a candidate appears or submits his credentials. Sec., Dr. Benedict Cooper, Agana.

PUERTO RICO: *Examination.* San Juan, Sept. 7-11. Sec., Dr. Joaquin Mercado Cruz, Box 9156, Santurce.

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

ALASKA: On application. Juneau or other towns in Territory as decided by Board. *Reciprocity.* On application. Sec., Dr. C. Earl Albrecht, Box 1931, Juneau.

ARKANSAS: *Examination.* Little Rock, Oct. 5-6. Sec., Dr. Louis E. Gebauer, 1002 Donaghey Bldg., Little Rock.

COLORADO: *Examination.* Denver, Sept. 8-9. Sec., Dr. Esther B. Starks, 1459 Ogden St., Denver 18.

MICHIGAN: *Examination.* Detroit and Ann Arbor, Oct. 8-9. Sec., Mrs. Anne Baker, Mason Bldg., Lansing 2.

NEBRASKA: *Examination.* Omaha, Oct. 5-6. Director, Mr. Husted K. Watson, Room 1009, State Capitol Bldg., Lincoln 9.

OREGON: *Examination.* Portland, Sept. 11 and Dec. 4. Sec., Mr. Charles D. Byrne, State Board of Higher Education, Eugene.

TENNESSEE: *Examination.* Memphis, Sept. 20-21. Sec., Dr. O. W. Hyman, 874 Union Ave., Memphis 3.

TEXAS: *Examination.* Austin, Oct. 22-23. Address, Mrs. Betty Ratcliff, Chief Clerk, 407 Perry-Brooks Bldg., Austin.

WISCONSIN: *Examination.* Madison, Sept. 24. Milwaukee, Dec. 4. Sec., Mr. W. H. Barber, 621 Ransom St., Ripon.

*Basic Science Certificate required.

DEATHS

Haviland, Frank Ross ⊕ Syracuse, N. Y.; born Jan. 18, 1880; Syracuse University College of Medicine, New York, 1903; formerly on the faculty of Columbia University College of Physicians and Surgeons in New York, where he was clinical professor of psychiatry at Fordham University School of Medicine; at one time clinical professor of neurology at the Long Island College Hospital in Brooklyn; life fellow of the American Psychiatric Association; life member of the New York Society for Clinical Psychiatry; member of the National Association for Mental Health; emeritus member, Syracuse Mental Hygiene Committee and the Syracuse Academy of Medicine; from 1942 to 1946 neuropsychiatrist with an armed forces induction team, for which he received the Selective Service Medal; consulting psychiatrist at the Marcy (N. Y.) State Hospital and the Syracuse Psychopathic Hospital; at one time on the staff of the Brooklyn State Hospital; served as director of psychiatry at the Manhattan State Hospital in New York City; died June 27, aged 74, of emphysema.

Egdahl, Anfin ⊕ Rockford, Ill.; born in Menomonie, Wis., Dec. 25, 1875; Johns Hopkins University School of Medicine, Baltimore, 1904; member of the American Trudeau Society; past president of the Winnebago County Medical Society and the Winnebago County Tuberculosis Association; at one time on the faculty of the State University of Iowa College of Medicine in Iowa City; since 1946 county medical examiner; served during World War I; later joined the reserves, serving as commanding officer of the 73rd evacuation hospital; held the rank of colonel when he was transferred to the inactive reserve; at one time head of the department of preventive medicine at the University of North Dakota School of Medicine in Grank Forks, and director of public health of the state of North Dakota; chairman, board of trustees of the Rockford Municipal Sanatorium; died July 6, aged 78, of coronary occlusion.

Kibler, Clarence LeRoy ⊕ Columbia, S. C.; born July 16, 1873; Maryland Medical College, Baltimore, 1907; past president of the Columbia Medical Society and vice-president of the South Carolina State Medical Association; past president of the Seaboard Air Line Railroad Surgeons' Association; formerly member of the board of trustees of Newberry College; during World War I a member of the regional board of examiners; for many years state supervisor in ophthalmology for the department of public welfare and vice-president of the Union National Bank, which he helped organize; member of the medical staffs of Columbia and Providence hospitals and of Baptist Hospital, where he died June 19, aged 80.

Schlesinger, Lee Cahn ⊕ New Orleans; born in Memphis, Tenn., March 29, 1912; University of Tennessee College of Medicine, Memphis, 1935; specialist certified by the American Board of Orthopaedic Surgery; assistant professor of clinical orthopedics at Tulane University of Louisiana School of Medicine; member of the American Academy of Orthopaedic Surgeons; fellow of the American College of Surgeons; served during World War II; consultant in orthopedic surgery at Bogalusa (La.) Medical Center; affiliated with Charity Hospital and Touro Infirmary; consultant in orthopedics at Flint-Goodridge Hospital; orthopedic surgeon for Louisiana State Crippled Childrens Program; consulting orthopedic surgeon for the Louisville and Nashville Railroad; died June 27, aged 42.

Blesse, Frederick A., Brigadier General, U. S. Army, retired, Richmond, Va.; born in Elgin, Ill., Nov. 22, 1888; the Hahnemann Medical College and Hospital, Chicago, 1913; entered the Medical Corps of the U. S. Army as a first lieutenant in January, 1918; promoted through the various ranks to that of colonel, July, 1943; promoted to brigadier general in April, 1948; retired Nov. 30, 1948, remaining on active duty until Nov. 30, 1950; served as chief surgeon in North Africa during World War II and for three years with the Army Ground Forces in Fort Monroe; held the Distinguished Service Medal and the Legion of Merit; since December, 1950, director of the Henrico County Health Department; died June 4, aged 65, of coronary thrombosis.

Fellows, Ralph Manos ⊕ Wauwatosa, Wis.; born in Salisbury, Mo., Aug. 28, 1894; University and Bellevue Hospital Medical College, New York, 1922; specialist certified by the American Board of Psychiatry and Neurology; member of the American Psychiatric Association and the Central Neuropsychiatric Association; fellow of the American College of Physicians; served during World Wars I and II; at one time vice-president of the Kansas Society for Mental Hygiene; resigned in 1940 as superintendent of Osawatomie (Kan.) State Hospital; formerly clinical director of the Menninger Clinic in Topeka, Kan.; medical director of the Milwaukee County Asylum for Chronic Insane; died in Milwaukee June 20, aged 59, of heart disease and hypertension.

Nelson, Moses Russell, New York City; born in Radnor, Pa., March 21, 1895; University of Pennsylvania School of Medicine, Philadelphia, 1920; assistant clinical professor of obstetrics and gynecology at the New York Medical College, Flower and Fifth Avenue Hospitals; fellow of the International College of Surgeons, Harlem Surgical Society, American Academy of Science, American Museum of Natural History, and the American College of Surgeons; member of the National Medical Association; consultant at Community Hospital in Newark, N. J.; affiliated with the New York City Hospital as associate visiting obstetrician and gynecologist; died June 24, aged 58, of cerebral hemorrhage.

Gamble, Hugh Agnew ⊕ Greenville, Miss.; born in Saltillo, Aug. 19, 1876; Medical Department of Tulane University of Louisiana, New Orleans, 1904; member of the House of Delegates of the American Medical Association from 1930 through 1932 and in 1937; member of the Southern Surgical Association and the Southeastern Surgical Congress; fellow of the American College of Surgeons; member of the founders group of the American Board of Surgery; past president of the Mississippi State Medical Association, Mississippi State Hospital Association, and the Delta Medical Society; city councilman; on the staff of the King's Daughters Hospital, where he died July 11, aged 77, of cancer.

Rafsky, Henry Aaron ⊕ New York City; University and Bellevue Hospital Medical College, New York, 1913; specialist certified by the American Board of Internal Medicine; member of the American Gastro-Enterological Association and the National Gastroenterological Association; fellow of the American College of Physicians; clinical professor of medicine (gastroenterology) New York Post-Graduate Medical School and Hospital, Columbia University; president of the medical board of the Home and Hospital of the Daughters of Jacob; on the staff of the Lenox Hill Hospital; died in Atlantic Beach, N. Y., July 31, aged 64.

Cress, Orlando Elsworth ⊕ Van Wert, Ohio; Eclectic Medical College, Cincinnati, 1921; served as county coroner; veteran of World Wars I and II; on the staff of the Van Wert County Hospital; died June 19, aged 65, of coronary occlusion.

Crossen, Henry Francis ⊕ Detroit; Indiana University School of Medicine, Indianapolis, 1919; specialist certified by the American Board of Otolaryngology; fellow of the American College of Surgeons; affiliated with Harper, St. Joseph's Mercy, and Mount Carmel Mercy hospitals; senior otologist at the Detroit House of Correction; died June 20, aged 60, of coronary thrombosis.

Doyle, Leo Walter Jr., San Francisco; Northwestern University Medical School, Chicago, 1943; at one time assistant clinical professor of obstetrics and gynecology at the University of California Medical School; served during World War II; died June 28, aged 35, of acute hemorrhagic pancreatitis.

⊕ Indicates Member of the American Medical Association.

Dunigan, Jay Thomas ⊕ Albany, N. Y.; Albany Medical College, 1934; served during World War II; physician for the city public schools; on the staff of St. Peter's Hospital; died in Portland, Me., July 7, aged 43.

Eastman, Claude Washington, Carlin, Nev.; Washington University School of Medicine, St. Louis, 1904; for many years district surgeon for the Southern Pacific Railroad; died in Elko (Nev.) General Hospital June 24, aged 80, of uremia and hypostatic pneumonia.

Eldridge, Jesse Cleveland, Chattanooga, Tenn.; Vanderbilt University School of Medicine, Nashville, 1913; served during World War I; at one time health director of Hamilton County; died in St. Petersburg, Fla., June 11, aged 67, of bronchopneumonia.

English, Merton Alden, Chevy Chase, Md.; George Washington University School of Medicine, Washington, D. C., 1915; died July 25, aged 75.

Fender, Marion Sims, Ehrhardt, S. C.; Emory University School of Medicine, Atlanta, 1915; also a druggist; for many years chairman and member of the board of trustees of the school district; died in the Columbia Hospital June 17, aged 63, of cerebral thrombosis.

Fulton, William Van Voorhis ⊕ Everett, Wash.; Jefferson Medical College of Philadelphia, 1912; died in Everett General Hospital July 5, aged 71, of acute myocardial infarction.

Gay, Clarence Bertram ⊕ Vineyard Haven, Mass.; University of Pennsylvania Department of Medicine, Philadelphia, 1899; formerly supervising censor, executive councilor, and president of the Worcester North District Medical Society; died in Martha's Vineyard Hospital in Oak Bluffs April 30, aged 81, of arteriosclerotic heart disease.

Goldman, Max ⊕ Kansas City, Mo.; Kansas City Medical College, 1901; past president of the Jackson County Medical Society; fellow of the American College of Surgeons; affiliated with Menorah and St. Mary's hospitals; died July 13, aged 73, of cardiac disease.

Goodloe, Hart, Tallahassee, Fla.; University of Louisville (Ky.) Medical Department, 1900; served during World War I; died in Biloxi, Miss., April 21, aged 79, of myocardial infarction and coronary occlusion.

Goodrich, Elmer Everett ⊕ Holdenville, Okla.; Barnes Medical College, St. Louis, 1902; served during World War I; formerly practiced in Chickasha, where he was director of the county-city health unit; died June 18, aged 79.

Grainger, Gustavius A. ⊕ Farmington, W. Va.; Vanderbilt University School of Medicine, Nashville, Tenn., 1904; died May 4, aged 71, of cardiac decompensation.

Groman, Herman Charles ⊕ Hammond, Ind.; Rush Medical College, Chicago, 1907; formerly health commissioner of Hammond; died in White Lake, Mich., July 21, aged 72.

Halleck, Frank Dana ⊕ Bowling Green, Ohio; Ohio Medical University, Columbus, 1898; past president of the Wood County Medical Society; died in Wood County Hospital May 14, aged 81, as the result of a fall.

Herskowitz, Max, New York City; Long Island College Hospital, Brooklyn, 1918; died July 2, aged 57, of heart disease.

Hollingsworth, Marshall P. ⊕ Princeton, Ind.; Medical College of Indiana, Indianapolis, 1891; formerly county coroner; medical examiner for the draft board during World Wars I and II; at one time medical examiner for the pension board; for many years medical examiner for the Metropolitan Life Insurance Company and the Southern Railway; died June 16, aged 92, of cerebral thrombosis.

Howell, Samuel Monroe ⊕ Cartersville, Ga.; Atlanta School of Medicine, 1913; served during World War I; founder of the Howell-Quillian Clinic-Hospital; died April 13, aged 64, of carcinoma of the bladder.

Huffman, Logan Herbert, Oklahoma City; University of the South Medical Department, Sewanee, Tenn., 1903; served during World War I; died in St. Anthony's Hospital June 19, aged 81.

James, Byrd, Chicago; Meharry Medical College, Nashville, Tenn., 1915; died in the Provident Hospital July 15, aged 73, of bronchopneumonia and carcinoma of the esophagus.

James, Charles Stanton ⊕ Omaha; Omaha Medical College, 1899; University and Bellevue Hospital Medical College, New York, 1900; fellow of the American College of Surgeons; affiliated with St. Catherine's Hospital and Doctors Hospital, where he died May 29, aged 76, of carcinoma of the stomach.

Jenkins, Harry Loveless ⊕ Arcata, Calif.; University of California Medical School, San Francisco, 1925; member of the American Academy of General Practice; served during World War I; died in the Trinity Hospital July 6, aged 57.

Johnson, Walter Brenaman, Cambridge, Md.; University of Maryland School of Medicine and College of Physicians and Surgeons, Baltimore, 1928; secretary-treasurer of the Dorchester County Medical Society; county and city health officer; while in Cumberland, served as city and county health officer; formerly health officer of Caroline County at Denton; president of the Rotary Club; on the staff of the Cambridge Hospital; died June 4, aged 52, of cerebral hemorrhage.

Jones, John M. ⊕ Millersburg, Ohio; Starling Medical College, Columbus, 1895; in 1917 appointed chief medical examiner for Holmes County Draft Board; died July 6, aged 83.

Kincheloe, John E. ⊕ Hardinsburg, Ky.; Kentucky School of Medicine, Louisville, 1899; died June 25, aged 76, of coronary thrombosis.

King, Ossian Homer ⊕ Hot Springs National Park, Ark.; College of Physicians and Surgeons, Little Rock, 1911; specialist certified by the American Board of Ophthalmology; member of the American Academy of Ophthalmology and Otolaryngology; past president of the Garland County Medical Society; on the staff of St. Joseph's Hospital; died in Fort Roots Hospital, North Little Rock, June 15, aged 67, of lower nephron nephrosis, thrombosis, and arteriosclerosis.

Knoop, William A. ⊕ Chesterfield, Ill.; Northwestern University Medical School, Chicago, 1905; died in Carlinville July 6, aged 73, of arteriosclerotic heart disease.

Lee, Elbert Johnson Jr. ⊕ St. Louis; Barnes Medical College, St. Louis, 1898; served during World War I; at one time associated with the U. S. Public Health Reserve; member of the state legislature, 1943-1944; formerly superintendent of the Charles B. Towns Hospital in New York, City Sanatorium, and City Hospital; served on the staffs of the Barnes Hospital and the Missouri Baptist Hospital, where he died June 20, aged 77, of coronary thrombosis.

Leland, John Tyndan ⊕ Mill Valley, Calif.; the Hahnemann Medical College and Hospital, Chicago, 1899; died suddenly July 20, aged 80, of cerebral hemorrhage.

Lichtenstein, Perry Maurice ⊕ New York City; Cornell University Medical College, New York, 1910; also a lawyer; for many years resident physician in Tombs Prison; medical assistant to district attorney; author of "A Doctor Studies Crime"; died June 14, aged 67.

Lukins, Aaron Tomlin ⊕ Tacoma, Wash.; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1910; served with the British Expeditionary Force overseas during World War I; past president of the Whitman County Medical Society and Lewis County Medical Society; died April 14, aged 71, of cerebral thrombosis and arteriosclerosis.

McAfee, James D., Cleveland; Homeopathic Hospital College, Cleveland, 1891; past president of the old city board of health; superintendent of the Cleveland City Hospital from 1906 to 1913; died June 26, aged 92, of coronary occlusion.

McCleery, Thomas Sandrock ⊕ Hazel Crest, Ill.; Chicago Medical School, 1936; health officer; on the staff of the Hazel Crest General Hospital, and the Illinois Central Hospital in Chicago, where he died July 26, aged 55, of ruptured dissecting aneurysm.

McClelland, Willis Barr ⊕ Franklin, Pa.; Boston University School of Medicine, 1931; served during World War II; member of the American Trudeau Society; formerly affiliated with the Pennsylvania State Sanatorium for Tuberculosis in Hamburg; died June 6, aged 48.

McDiarmid, Thomas Scott ⊕ Gadsden, Ala.; Birmingham Medical College, 1909; served during World War I; died July 4, aged 70, of carcinoma of the lung.

McEvoy, Frank Edward ⊕ Providence, R. I.; University of Pennsylvania School of Medicine, Philadelphia, 1913; specialist certified by the American Board of Surgery; member of the New England Surgical Society; past president of the Providence Surgical Society; fellow of the American College of Surgeons; affiliated with State Hospital for Mental Diseases in Howard and the Roger Williams General and St. Joseph's hospitals; died July 15, aged 66, of carcinoma of the prostate.

Mainegra, Robert James, New Orleans; Tulane University of Louisiana School of Medicine, New Orleans, 1906; died June 14, aged 71.

Maker, Ralph Waldo, Acampo, Calif.; College of Medical Evangelists, Loma Linda and Los Angeles, 1918; for many years on the staff of the Glendale (Calif.) Sanitarium and Hospital and the Norwalk (Conn.) State Hospital; on the staff of the Stockton (Calif.) Hospital; died July 9, aged 70, of cerebral embolism.

Malmstone, Francis A. ⊕ Griffith, Ind.; Chicago College of Medicine and Surgery, 1914; on the staffs of St. Margaret Hospital in Hammond, St. Catherine's, Methodist, and St. Mary's Mercy hospitals in Gary; died in the American Hospital in Paris, France, July 2, aged 77, of cardiac thrombosis.

Martin, Robert Alfonzo ⊕ Pell City, Ala.; Vanderbilt University School of Medicine, Nashville, 1901; on Jan. 8, 1953, the Greater Pell City Chamber of Commerce sponsored a "Dr. Martin Day" honoring him for the 50 years of medical service and friendship that he had given this community; medical director and owner of the Pell City Infirmary; died in the Ochsner Foundation Hospital, New Orleans, July 10, aged 74, of coronary thrombosis.

Moss, John Gaines ⊕ Johnson City, Tenn.; Chattanooga Medical College, 1901; member of the Southeastern Surgical Congress; fellow of the American College of Surgeons; served in France during World War I; affiliated with the Memorial Hospital; died June 18, aged 75.

Mullikin, Clarence William ⊕ Greensburg, Ind.; Indiana University School of Medicine, Indianapolis, 1908; died in Tucson, Ariz., April 30, aged 73, of peritonitis, paralytic ileus, acute obstructive cholecystitis, and fatty degeneration of the liver.

Nahigan, Matthew Nigohos, Los Angeles; Yale University School of Medicine, New Haven, 1907; affiliated with St. Vincent Hospital, where he died June 26, aged 77, of heart disease.

Natherson, Frank Marion ⊕ Parma, Ohio; Ohio State University College of Medicine, Columbus, 1934; affiliated with Evangelical Deaconess Hospital in Cleveland; died June 19, aged 46, of coronary thrombosis.

Nathorff, Erich ⊕ New York City; Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin, Prussia, Germany, 1914; co-author of several standard German textbooks; died in Atlantic City, N. J., June 25, aged 64, of coronary thrombosis.

Neil, James Benton ⊕ Knoxville, Tenn.; Vanderbilt University School of Medicine, Nashville, 1912; died in Fort Sanders Hospital June 21, aged 66, of endocarditis.

Newton, Charles Irving ⊕ Geneseo, N. Y.; University of Michigan Homeopathic Medical School, Ann Arbor, 1907; died July 7, aged 70.

O'Neil, Dillon Garrett ⊕ Chicago; Georgetown University School of Medicine, Washington, D. C., 1913; served during World War I; died in the Veterans Administration Hospital in Hines, Ill., July 31, aged 67, of bilateral bronchopneumonia.

Paulus, Clarence Allen ⊕ Telford, Pa.; Medico-Chirurgical College of Philadelphia, 1915; served during World War I; affiliated with Grand View Hospital in Sellersville, where he died May 30, aged 68, of ruptured aneurysm of the abdominal aorta.

Peiffer, Charles Oscar, Philadelphia; Jefferson Medical College of Philadelphia, 1904; associated on the staffs of Episcopal Hospital and Stetson Hospital; died June 29, aged 76, of arteriosclerosis and renal disease.

Powers, Arthur Linn, Locke, N. Y.; University of the City of New York Medical Department, 1890; died July 2, aged 88.

Rachlin, William ⊕ Brooklyn; New York University Medical College, New York, 1898; consulting physician at the Maimonides Hospital; died in Manhasset July 21, aged 79, of acute uremia.

Randall, Emma Jean Pearson, Van Wert, Ohio; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1899; died in Toledo, July 10, aged 79, of cancer.

Reed, John Chamberlain ⊕ Duquesne, Pa.; Western University of Pennsylvania Medical Department, Pittsburgh, 1907; died in Sharon May 23, aged 76, of pulmonary embolus and arteriosclerotic heart disease.

Renfrow, Thomas Franklin ⊕ Billings, Okla.; Missouri Medical College, St. Louis, 1896; died in Fort Roots Veterans Administration Hospital in North Little Rock July 8, aged 82, of cerebral thrombosis and arteriosclerosis.

Riemer, Hugo Bruno Charles ⊕ Norwood, Mass.; Harvard Medical School, Boston, 1904; specialist certified by the American Board of Ophthalmology; member of the American Ophthalmological Society and the New England Ophthalmological Society; at one time associated with the U. S. Public Health Service; consultant at the Massachusetts Hospital School in Canton, Wrentham (Mass.) State School; consultant in surgery at the Massachusetts Eye and Ear Infirmary in Boston; died July 10, aged 78, of cerebral hemorrhage.

Salters, Leland Blackwood ⊕ Florence, S. C.; University of Maryland School of Medicine, Baltimore, 1903; affiliated with Bruce and Saunders hospitals; died May 28, aged 76, of myocarditis.

Saltus, Lloyd Seaman ⊕ Brookside, N. J.; Columbia University College of Physicians and Surgeons, New York, 1935; served during World War II; affiliated with Columbia-Presbyterian Medical Center in New York and the Morristown (N. J.) Memorial Hospital; died June 19, aged 45, of cancer of the brain.

Simon, Peter Joseph ⊕ Cleveland; Eclectic Medical College, Cincinnati, 1938; died in the Deaconess Hospital, Cleveland, June 7, aged 43, of coronary sclerosis.

Thomas, Calvin Merrill, Brewer, Maine; University of Pennsylvania Department of Medicine, Philadelphia, 1902; past president of the Penobscot County Medical Society; affiliated with the Eastern Maine General Hospital in Bangor, where he died May 22, aged 74, of carcinoma of the prostate with metastasis.

Thompson, William Givens ⊕ Holden, Mo.; Missouri Medical College, St. Louis, 1890; specialist certified by the American Board of Radiology; served during World War I; for many years mayor of Holden; died July 2, aged 72, of auricular fibrillation.

Webb, Floyd ⊕ Blytheville, Ark.; Memphis (Tenn.) Hospital Medical College, 1913; served during World War I; died in Chickasawba Hospital May 28, aged 65.

Welfield, Samuel Estin ⊕ San Francisco; College of Physicians and Surgeons of San Francisco, 1918; member of the Industrial Medical Association; died June 28, aged 65.

DIED WHILE IN MILITARY SERVICE

Manning, Joseph Crockett Jr. ⊕ Indianapolis; born in St. Joseph, Mo., May 29, 1916; University of Tennessee College of Medicine, Memphis, 1942; fellow of the American College of Surgeons; interned at the Methodist Hospital, where he was on the associate staff; served a residency at the Indiana University Medical Center; on the staff of the Indianapolis General Hospital; joined the U. S. Air Force Reserve in March, 1953, serving as a captain, in charge of surgery at Tinker Air Force Base in Oklahoma City; died in Sheppard Air Force Base Hospital in Wichita Falls, Texas, April 3, aged 37, of bronchogenic carcinoma.

FOREIGN LETTERS

BELGIUM

Cancer of the Rectum.—At the Annual Meeting of the Belgian Surgical Association in May, 1954, in Brussels, Daumerie and Samain reported that primary or secondary cancer of the rectum is an epithelioma of which the exact location is of great importance because its malignancy is in inverse proportion to the age of the patient. Patients with cancer of the rectum are usually seen by the surgeon much too late. A fundamental principle upheld by Daumerie and Samain is that resection of the rectum must be wide and that sphincter preservation must be sacrificed to adequate resection. Surgery that aims to be curative is a major operation and should only be used on patients who can tolerate the risks attendant on such intervention. Resection is indicated when the tumor is inaccessible to rectal palpation or only its lower border can be felt. It is useless to try to preserve the anal canal if the cancer is felt at 5 cm. from the anus. Amputation is preferable to resection because, in resection, there is a greater risk of recurrence. Surgeons who favor resection at any cost must not forget that continence does not necessarily follow sphincter preservation. Resection may be performed abdominally, abdominotransanally, or abdominosacrally. The only choice to be made in amputation lies in the location of the artificial anus (iliac anus or perineal anus). Cure of the rectal cancer is worth the inconvenience of an iliac anus, which, though definitely a major deformity, is practicable and allows better control than the perineal anus. The surgical treatment of cancer of the rectum is satisfactory in 60 to 80% of patients. With the use of antibiotics and the discontinuation of anastomoses by combined routes, the operative mortality has reached a low level. The improvement in long-term results stems from the abandonment of attempts to preserve the sphincter and from earlier diagnosis, resulting in intervention. Further progress will probably result from improved methods of diagnosis rather than from such surgical innovations as pelvic evisceration.

Consumption of Alcohol.—The *Revue de l'alcoolisme* of January-March, 1954, showed that in 1948 each Belgian consumed an average of 4.77 liters of wine and each Frenchman 150 liters. The annual consumption of beer at that time in Belgium was 133 liters per capita. This represents a decrease from 1931 when the annual consumption was over 200 liters per capita, but this tendency to decrease is compensated for by the present increase in the alcoholic content. According to these figures, the Belgians have the greatest per capita consumption of beer in the world, followed by the United States with 67.1 liters and the United Kingdom with 61.4 liters. The consumption in France is only 20.1 liters. In Belgium, the annual consumption of distilled liquors was 1.42 liters per capita in 1950 as compared with that of France (5.92 liters), Sweden (5.2 liters), and the United States (2.5 liters). On Jan. 1, 1950, Belgium had 82,666 cafes, restaurants, and hotels. Adding the 35,810 wine and spirit shops where drinking is not allowed on the premises, there is one place for every 63 inhabitants where it is possible to obtain liquor.

Disappearance of Malaria.—At the Fifth Session of Comparative Pathology in Strasbourg, Wanson stated that malaria disappeared in Belgium about 1870 despite the persistence of Anopheles maculipennis atroparvus, the principal vector in this disease. The disappearance of malaria was due to the use of damming systems to separate salt and fresh water (organization of the Wateringes), agricultural progress in the suppression of polders and the consequent reduction in anopheline vectors, an increase in the livestock preferred by anopheline mosquitos, and the continued use of quinine in the treatment of intermittent fevers.

Bovine Tuberculosis in Human Beings.—The Belgian Society for Scientific Studies on Tuberculosis invited several French and Swiss authorities to discuss the importance of the bovine tubercle bacillus in human lesions. Clinicians tend to believe too readily in the presence of bovine tubercle bacilli in the lesions they treat. In France, definite evidence of bovine tubercle bacilli was found in only 2 to 5% of tuberculous patients. Many cervical and mediastinal lymph node infections are caused by human tubercle bacilli. Since 1939 human infections with bovine tubercle bacilli have been greatly reduced by the removal of contaminated livestock. Human tuberculosis caused by the bovine bacillus can be confirmed only by isolation of the causative organism and its identification by its antigenic properties. The disease is seen oftenest in children under 5 years of age. The incidence of this type of tuberculosis is relatively high in England, Scotland, and Switzerland, less in Canada, insignificant in France, and nil in the United States. In all countries, infection by the bovine bacillus is commoner in rural than in urban areas. The prevention of human tuberculosis from bovine bacilli consists of prohibiting the consumption of unpasteurized milk and combatting bovine tuberculosis.

Physicians' National Service.—The Belgian Medical Federation has organized a physicians' national service that will assure medical care to all inhabitants of the kingdom whose incomes are lower than a certain amount. The medical fees will be determined for each region. All physicians are asked to participate in this service. The service will publish a list of the physicians who will participate to an extent that is either fixed or increases by a certain percentage, together with the hours and other conditions under which this care will be given. All laboring persons will thus be able to consult the physician of their choice at fixed fees and be assured of a completely free choice. This service was set up under the presidency of Dr. Craps of Brussels and the patronage of Professors Bordet, Van Genuchten, Dermon, and Le Platt.

Fluoridation of Drinking Water.—Hianne reported to the Belgian Hygiene and Social Medicine Association that, because the ingestion of small amounts of fluorine in childhood constitutes prophylaxis against tooth decay, the water supply of Namur, all of which comes from the same well, will be treated with sodium fluoride. The school children of this community will undergo regular radiographic examinations of their deciduous teeth and their six year molars. The roentgenograms will be compared with those taken in the schools of a neighboring community whose water does not contain fluorine.

BRAZIL

Antimalaria Campaign.—The results of the epidemiological survey carried out in 1947, shortly after the organization of the malaria division of the National Department of Health, and continued during 1948, showed that malaria was present in 67.2% of the 1,780 counties in Brazil, in most of them endemically and in the rest under the form of periodic epidemic outbreaks of varying severity. The endemic and epidemic manifestations of the disease spread over coastal districts along the 3,800 miles of the Atlantic seaboard and the valleys of the great and small rivers. Rio Grande do Sul (a southern state having the coolest climate), the semiarid inland districts of the northeastern states, most of the Paraiba river valley in the state of Rio de Janeiro, and small isolated areas in several parts of the country were free from malaria. According to conservative estimates, a minimum of 6 million persons were affected by malaria every year (12,180 per 100,000 population in 1947), with about 65,000 deaths directly attributable to the disease (132 per 100,000). Before the founding of the malaria division, the antimalarial activities of the federal government were almost limited to the cooperation with state authorities in stamping out the most important outbreaks of the disease. Even before the end of the epidemiological survey, effective measures were begun against adult mosquitoes, consisting of house spraying with chloro-

The items in these letters are contributed by regular correspondents in the various foreign countries.

phenothane (DDT) in the São Francisco river valley (states of Bahia, Sergipe, and Alagoas) and in the entire state of Rio de Janeiro, which surrounds the Federal District. The results of this experiment led to a rapid expansion of the program, which reached its highest point in 1950, when more than 2.6 million dwellings were sprayed in 836 counties covering an area of about 2,650,000 square miles (78% of the whole country) with a population of 25,311,000 (more than 48% of the population in that year). This house spraying was continued in 1951, 1952, and 1953.

A full report has been published by the Malaria Division. In the areas regularly sprayed with chlorophenothane, the transmission of malaria may fall to levels so low that the mere determination of hemoscopic indexes in samples will not give a true picture of the situation. It is important to maintain a routine search for suspected cases of the disease and to confirm the diagnosis through blood examination. In the large areas protected by chlorophenothane the incidence of malaria showed a reduction of about 95%. Better results were obtained, whatever the species of the vector, when two cycles of spraying were carried out in the year, as was done in seaboard districts, where the reduction reached 98 or 99%. In these districts where the transmission is mainly due to Anopheles tarsimaculatus, Anopheles darlingi, and Anopheles albitarsis, the houses alone were sprayed. In some inland districts, however, where cases of malaria still occurred, such factors as (1) extradomiciliary transmission by A. darlingi, even with two cycles of chlorophenothane house spraying in the year; (2) transmission by A. darlingi in areas where the houses have very irregular walls or no walls at all, hampering the protection from chlorophenothane; and (3) extradomiciliary and peridomiciliary transmission by Anopheles cruzi and Anopheles bellator in southern coastal districts could not yet be entirely eliminated. Cases of malaria occurred in houses internally sprayed with chlorophenothane where infected specimens of A. darlingi and A. bellator were captured on the external surfaces of the walls and on trees and bushes. As a contribution to the control of residual malaria in such difficult situations, Dr. Mario Pinotti, director of the Malaria Division, suggested the use of chloroquine mixed with table salt. The method was used in several districts in 1953 with encouraging results, the parasitoscopic index falling to 0.3, 0.1, and even 0%, in contrast with values of 6 to 25% in the same districts before the generalized use of chloroquine and salt or in similar areas at the same time where the method was not in use.

Vital Statistics for Rio de Janeiro.—Rio de Janeiro is a fast growing city. In 1940 it had 1,764,141 inhabitants, and in 1950 it had 2,413,152. The midyear population for 1954 has been computed to be nearly 2,700,000. The data for 1953, published in the bulletin of the Federal Bureau of Vital Statistics, show that the annual death rate from all causes declined from an average of 15.5 per thousand population for the five year period 1944 to 1948 to 11.7 in 1953. On the other hand, the annual birth rate increased from an average of 20.8 per thousand to 26.1 for the same periods. Computing a ratio between the total number of live births and the number of deaths from all causes, we get a vital index whose value increased from 1.33 for the period 1944 to 1948 to 2.23 for 1953. The infant mortality rate for 1944 to 1948 was 128 deaths per thousand live births, and for 1953, 109. The maternal mortality, which was 5.5 puerperal deaths per thousand live births for the period 1944 to 1948, was 3.5 in 1953. The greater part of this reduction was due to the decline in puerperal infection that resulted from the increasing use of antibiotics. Deaths from tuberculosis of all forms declined from 295 per 100,000 population in the period 1944 to 1948 to 94 in 1953. Deaths from typhoid fever fell from 7.9 per 100,000 to 1.5 in the same periods. The corresponding figures for the several forms of dysentery were 8 deaths per 100,000 and 5.5. Deaths from whooping cough dropped from 7.4 per 100,000 to 2.6; from measles increased from 2.8 to 5.2; and from diphtheria decreased from 4.3 to 3.4. Deaths from poliomyelitis, which formerly had been relatively rare with annual rates of about 0.2 per 100,000, increased to 1.4 in 1953; deaths from heart diseases increased from 158 per 100,000 to 216; cancer, from 18.2 per 100,000 to 23.6; and motor vehicle accidents, from 68.1 per 100,000 to 83.1. Deaths from diabetes remained almost unchanged, fluctuating between 6.8 and 8.9 per 100,000. The death rate from appendicitis showed a remarkable reduction, from an average of 5 deaths per 100,000 in the period 1944 to 1948 to 1.9 in 1953.

ENGLAND

Another Report on General Practice.—In December, 1950, the Central Health Services Council appointed a committee "to . . . report on whether the existing arrangements for engaging in general practice under the National Health Service are such as to enable general medical practitioners to provide the best possible standard of service." The committee, made up predominantly of medical men and including consultants and general practitioners, has now presented its report. This starts with the categorical statement that "general practice . . . is fundamental to the best practice of medicine and to the best interests of patients. It cannot be replaced by a congeries of specialisms." On July 1, 1952, the latest date for which full figures are available, 20,162 physicians were engaged in general practice in the National Health Service in England and Wales. These included 17,204 persons providing "unrestricted" general medical service, 56% of whom were members of partnerships, the rest working alone. Provisional figures for 1953 suggest that the proportion in partnership has risen to over 60%, and this increasing cooperation between physicians is one of the most encouraging developments in the National Health Service.

In the committee's view the range of work of a general practitioner should normally include "(*a*) Continuing responsibility of the health of his patients. (*b*) The diagnosis, treatment and management of such illnesses as can properly be treated in the patient's home. (*c*) When the patient's condition requires it, reference to hospitals or specialists for diagnosis or treatment or both and at the same time the forwarding of the relevant details of such patients. (*d*) Emergency treatment. This includes the emergency treatment of any patient and is not confined to those on the doctor's list. (*e*) Supervision of the convalescent period and return to work. (*f*) The practice for his patients of preventive medicine and of health education. (*g*) The giving of certificates and other administrative and legal requirements, for example, notification of disease when statutorily required. *It would always assume* (*h*) Proper and sufficient arrangements for examining patients. (*i*) Clean, pleasant and adequately equipped accommodation. (*j*) Association with hospitals and local authorities."

Referring to the frequent assertions that since the introduction of the National Health Service there has been an increase in the number of items of service rendered by general practitioners to their patients, the committee suggests that "though this may be so, the increase has not been very great and has not been evenly distributed." Figures supplied by the registrar general indicate a steady increase in the proportion of consultations taking place in the physician's office as compared with those in the patient's home and in the number of women and children treated. The committee believes that "the arrangement whereby a family has, over a period of years, 'its own doctor' is an ideal one both for the doctor and the family, ensuring as it does continuity of treatment for the latter and affording the doctor valuable knowledge of the general background of the patient." At present about two-thirds of a physician's patients "may regard him as the family doctor, at least to the extent that father, mother and children in that family are all on the doctor's list." The committee "does not think that in general the advent of the National Health Service has disturbed the relationship between doctors and their patients . . . the relationship is good; in some, respects, indeed, it was found to be better than before, and this was attributed to the absence of the money bar and to increased cooperation between doctors."

In discussing the organization of general practice, the committee emphasizes the need for ancillary help in the practice and for an appointment system. "A doctor should not himself undertake what can be properly delegated to a nonmedical assistant. The provision of such help is easier to achieve in partnership and within groups of doctors because its cost can be shared. This is indeed an important advantage of group practice." Although the committee is enthusiastic about the

advantages of partnerships and group practice, it "does not share the view . . . that single-handed practitioners must as soon as possible be forced to link themselves up in formal groups." Of group practice, it says that normally the group should consist of three to six persons.

Among the difficulties encountered in establishing health centers under the National Health Service is the fact that the local authority is in control by virtue of being responsible, under the terms of the relevant act, for supplying them. "This at once causes misgivings in the minds of many general practitioners." Furthermore, once a practitioner has given up his private office for a suite in a health center, "he will have lost much of his independence because his patients may become attached to the centre and would not then, in the main, follow him back to other premises if he decided to break his connection with the centre." Conversely, many patients who have become used to visiting a physician at his own office might not be prepared to follow him to a health center and might transfer to a neighboring practitioner. The committee comments: "It could be argued that all these difficulties should be forcefully swept aside, but unfortunately they have their foundations in the hard facts of history and of intelligent self-interest. In practice, the doctor, as an independent contractor under the arrangements for general medical services, is having to gamble on the success of his step if he agrees to transfer his practice to a health centre. It is a speculative move in which he naturally requires assurances on charges, control, powers of dismissal and so on. . . . Many of the advantages both to doctors and to patients, which have in the past been urged in favour of health centres, may it is hoped be more easily secured through the evolution of group practices, consisting of doctors who have chosen to work together in communal premises which they own themselves."

In some practices, particularly in industrial areas, office accommodations are not all they should be. This is due in part to the postwar difficulties in any building program, no matter how small. Private building work and alternative accommodations are now available, and, therefore, it is not now unreasonable to urge improvements in office accommodations. The gross remuneration of general practitioners is at present calculated to include 38.7% for practice expenses, a large part of which is spent for office accommodations and equipment. "Only insistence of adequate standards can provide a proper safeguard against the otherwise paradoxical situation that the less a doctor spends on his practice expenses—which in many cases may mean the poorer his accommodations—the greater proportionately are his personal rewards from practice." Executive councils have the power to inspect office accommodations, and these councils are urged to make the necessary arrangements to ensure that the accommodations provided by physicians include (1) running water (or at least a wash basin with water), soap, and a towel in the consulting room, (2) a separate examining room or screened-off examining table, (3) a waiting room large enough to seat in reasonable comfort all who may need to wait, except in special circumstances such as a severe epidemic, (4) a bright and pleasant, well-lit, well-warmed, well-cleaned, and well-ventilated atmosphere, (5) furniture of reasonably good quality and domestic rather than institutional, and (6) access for patients to a toilet and wash basin.

The committee considers that a period of assistantship is normally the best introduction to general practice. The number of assistants employed by general practitioners in the National Health Service is around 2,000. It is admitted that there has been some abuse of the arrangements whereby physicians employ assistants and take on partners, and recommendations are made for removing these. One, for instance, is that in a partnership of two the agreement should normally provide for at least a third share for the new partner at the end of two or three years. The committee considered evidence in favor of methods of remuneration other than by capitation fees, namely, by salary, by sessional fees, or by fees for items of service, but came to the conclusion that "the present method by capitation fees is the most satisfactory. . . . Since it does not preclude the practitioner from doing other work outside general medical service under the National Health Service, it has also the advantage that it means that no special limit is set to the general practitioner's remuneration."

On the subject of the maternity medical service, the report states that "quite apart from the question of economy and expedience, the Committee thinks it is desirable that more normal confinements (except many primiparae and women who have had four or more children, amongst whom unforeseen abnormalities more commonly occur) should, if the housing and other relevant social conditions are satisfactory, take place in the patient's home." Of the confinements that take place in a hospital, it is considered that many could be dealt with by general practitioners if hospital boards would set aside hospital accommodation for this purpose. The committee could find no evidence that the provision of maternity medical services by general practitioners had decreased since the inception of the National Health Service.

The committee categorically rejected the repeated demand that a person, although electing to obtain medical treatment as a private patient, should nevertheless be allowed to obtain drugs under the National Health Service: "The Committee decided that it is right to regard prescribing as an integral part of general medical services and therefore does not recommend that doctors be allowed to use National Health Service prescription forms for their private patients." On the subject of private practice in general, the view is expressed that "the opportunity for private practice for doctors in the Service remains as part of the freedom both of the doctor and of the patient. This is, in the Committee's view, right." Most patients, however, have elected to come into the service, and physicians have accepted the full responsibility of ensuring that patients in the service are offered the highest standards of medical practice.

A strong plea is made for the wider acceptance of the suggestion that "general practitioners should have at their disposal hospital beds where they can retain responsibility for their patients, many of whom are now admitted to hospital, not primarily for medical reasons, but because home circumstances do not permit proper general, including nursing, attention. Consultant advice would be available when necessary." A plea is also made for the wider adoption of the system of clinical assistantships for general practitioners provided "the hospital should not regard the general practitioner who is a clinical assistant simply as a useful additional pair of hands." The committee also takes the view that radiological and pathological facilities should be directly available to practitioners. It reports that in 1952 nearly half the hospitals with radiological facilities made these available to general practitioners directly and carried out 741,000 examinations for them, an increase of 11% over 1951. Fifty-five per cent of pathology laboratories gave similar facilities—554,000 examinations in 1952, an increase of 21% compared with 1951.

Finally, on the subject of undergraduate education, the committee consider that "a student should be given as much opportunity to study the scope of general practice as he is to learn the scope, for example of surgery or preventive medicine." It is also recommended that selection committees for the admission of students to medical schools should include in their membership a physician engaged in, or with personal experience of, general practice.

Smoking and Cancer of the Lung.—There are several references to smoking and lung cancer in the annual report of the British Empire Cancer Campaign covering the year 1953. In introducing the report at the annual meeting, Prof. F. Dickens stated that the importance of cigarette smoking causing cancer of the lung is not yet known and that the situation is complicated by the fact that primary cancer of the lung occurs among nonsmokers as well as smokers. A further complication lies in the statistical association now found between the incidence of lung cancer and the density of population or its "degree of urbanization." Since campaign research workers have found that town air contains appreciable amounts of known chemical carcinogens, as well as other irritant substances, it is a reasonable hypothesis that the prolonged inhalation of such air, either alone or combined with tobacco smoking, may be conducive to pulmonary cancer.

At the London Hospital, R. H. Gwynn and M. N. Salaman have failed to produce tumors in mice from the weekly or twice weekly application to their backs over a period of 23 weeks of (1) a methanol extract of whole, unburnt cigarettes ("Virginian"

brand), (2) the same extract preceded by initial treatment with 9:10-dimethyl-1:2-benzanthracene, or (3) 0.1% purified nicotine in acetone.

At the University of Leeds a machine has been produced capable of smoking 36 cigarettes simultaneously at about the same rate and the same temperature as in man. Two extracts of cigarette smoke have been produced: (1) the condensate from the whole smoke dissolved in acetone and (2) the condensate dissolved in benzene, washed with acid (to remove base), alkali (to remove phenols), and water, and the solvent removed under pressure. This produced 18 gm. of a semisolid gum from 10,000 cigarettes. These extracts were tested in mice and rats for seven months, but no tumors appeared. Also at the University of Leeds, work has been carried out on the radioactivity of tobacco ash and smoke. The mean radioactivities per cigarette were equivalent to 25 mg., 28 mg., and 6 mcg. of potassium for the tobacco, ash, and smoke, respectively. The difference between the radioactivities of the tobacco and the ash are not significant. The report concludes that such radioactivity as is present in cigarette tobacco remains in the ash and that little, if any, is transferred to the smoke. Taking 6 mcg. as representing the maximum radioactivity (as potassium) that could be transferred to the lung by one cigarette and assuming that in the worst case this could lodge as a single particle, the dosage to cells adjacent to it can be shown to be of the order of 2 to 3 mr per week. This is about equal to the dosage that body cells receive continually from their average potassium content, local rays, and cosmic rays and is at least 10,000 times less than the dosage involved in cases where tumors have been known to be due to deposition of radioactive materials in the body.

In the cancer research department of the Royal Beatson Memorial Hospital, Glasgow, the treatment of CBA mice with commercial nicotine by skin application or orally did not result in an increased incidence of pulmonary tumors, though many animals survived more than two years of treatment. Application of a crude preparation of oxidized nicotine to the skin of 14 mice of a mixed stock gave adenomas of the lung in 7 animals. A comparison of chromatographic fractions of cigarette tobacco and of the acid extract of tobacco smoke showed that the stained chromatogram of the more volatile and soluble fractions of tobacco showed spots provisionally identified as nicotine and other related alkaloids, while similar fractions of the acid extract of tobacco smoke, apart from nicotine, contained less stainable material. The less soluble fraction of the tobacco smoke extract, on the other hand, contained almost as much stainable material as the corresponding tobacco extracts, although the rate of flow when developed on the chomatogram varied. The rate of flow of these spots and the color after staining with *p*-aminobenzoic acid appear to indicate the presence of oxidation products of nicotine and related alkaloids in these tobacco smoke fractions. Tobacco smoke tar and tobacco resin were also examined by paper partition chromatography, and the presence of nicotine was identified in both fractions.

Hospital and Community.—A detailed report of an investigation into various aspects of hospital care by Prof. T. Ferguson and Dr. A. N. MacPhail of the department of public health of the University of Glasgow has been published under the aegis of the Nuffield Provincial Hospitals Trust. Two separate but closely integrated studies are included in the report. One was of two "mixed" Scottish counties with a combined population of about 500,000 and having within the county boundaries some 5.7 hospital beds per 1,000 population (apart from beds for the treatment of mental illness) as well as easy access to city hospital sources. It was found that in these counties one person in every seven received hospital treatment—inpatient, outpatient or both—in the course of a year. Hospital morbidity was closely correlated with social and environmental conditions. Thus, respiratory disease, injuries, and disease of the skin and cellular tissues contributed more heavily to hospital load in the registrar general's social class 5 (unskilled laborers) than in the others. Conversely, duration of stay in a hospital in most disease groups was appreciably shorter among patients from social classes 1 and 2 (the executive and professional classes) than among patients from class 5.

The second study was of 705 men discharged after treatment in medical units in one of four hospitals for acute conditions in the west of Scotland. The clinicians in charge of these patients estimated that in just over 10% some preventable factor had made a major contribution to the breakdown responsible for admission to hospital. A follow-up showed that two years after discharge from the hospital, 171 had died and 60 could not be traced; the remaining 474 were seen in their homes. Of the 171 who had died, 119 were under the age of 65, and 70 were under the age of 55. Of the 474 interviewed, 111 were regarded as cured, 193 had continued to maintain the improvement achieved in the hospital, 106 had not improved and their medical condition was still unsatisfactory, and 64 were worse. In other words, two-thirds of those still alive two years after leaving the hospital had derived "substantial and lasting benefit" from hospital care. Over a period of seven years—five years before this study was instituted and two years after discharge from the hospital—118 of these 474 men had been admitted to the hospital on three or more occasions. Readmission was more frequent if home conditions were not good, and a relatively high proportion of the men who were not at work two years after leaving the hospital came from poor homes. Just under 20% of those who were at work at the end of two years were in unsuitable jobs. Since leaving the hospital, 106 of the 474 men had never worked.

The authors sum up as follows: "It is doubtful whether results substantially better than those presented in this report can be obtained with things as they are. Nor would mere multiplication of costly hospital beds within any reasonably practicable limits of itself effect great improvement; without more help in the transition from hospital to everyday life, without decent living conditions and reasonably suitable work to which the patients can return on leaving hospital, even the most careful and enlightened treatment is not likely to achieve a full measure of lasting benefit. It is important to preserve all that is best in our hospital tradition; but equally it is important to try to view hospital care in a true perspective."

Antimalarial Drugs.—The malaria subcommittee of the Colonial Medical Research Committee has published revised recommendations for the use of antimalarial drugs (*Lancet* **2**:1340, 1954). For causal prophylaxis they recommend chloroguanide (Proguanil) or pyrimethamine. The order of preference for suppressive drugs is chloroguanide, pyrimethamine, chloroquine, Amodiaquine, quinacrine (Atabrine), and quinine. A warning is given of the possibility of resistance to chloroguanide and pyrimethamine, especially if they are used indiscriminately as therapeutic agents. Cross resistance has been shown to exist between chloroguanide and pyrimethamine, so that if resistance to either becomes apparent a change of medicament to chloroquine, Amodiaquine, or quinacrine is indicated. For the treatment of an overt attack in nonimmune subjects, chloroquine or Amodiaquine is recommended. "Neither Proguanil nor pyrimethamine is sufficiently rapid in action to warrant their use in the treatment of malaria in nonimmune subjects." For the treatment of the overt attack in partially immune subjects such as the indigenous inhabitants of malarious districts, a single-dose treatment with chloroquine, chloroguanide, Amodiaquine, or quinacrine has proved effective. For emergency treatment, as in pernicious forms of malaria, quinine dihydrochloride intravenously, quinacrine methane sulphonate intramuscularly, or chloroquine intravenously or intramuscularly is indicated; oral administration should be substituted as soon as possible. For radical cure of vivax or malariae malaria, pamaquine naphthoate or primaquine should be used. During an overt attack a course of treatment with a schizontocidal drug must also be given. Careful supervision is necessary over patients taking any of the 8-aminoquinoline drugs, such as pamaquine or primaquine, because of the occasional unpredictable occurrence of acute intravascular hemolysis with or without hemoglobinuria. The 8-aminoquinolines are the only drugs that are able to destroy the sexual forms of Plasmodium falciparum in the peripheral blood. Chloroguanide and pyrimethamine have no demonstrable action on the gametocytes in the blood, but both have the property of preventing them from undergoing full development in the mosquito, so that mosquitoes feeding on patients taking either of these drugs are unable to transmit the disease to others.

Homologous Serum Jaundice—Several years ago a survey was instituted by the medical research council of the Ministry of Health and the department of health for Scotland with the object of comparing the incidence of homologous serum jaundice after administration of dried, irradiated plasma with that after administration of (1) dried, unirradiated, small-pool plasma, prepared concurrently by the usual methods in use since 1945, and (2) whole blood alone. The results are shown in the following table, taken from a preliminary report just published (*Lancet* **2**:1328, 1954).

Transfusion Fluid	No. of Patients: Treated with Transfusion	Jaundice of Doubtful Cause	Homologous Serum Jaundice Attributed to Transfusion	Deaths Attributed to Homologous Serum Jaundice
Blood	2,538	4	4 (0.16%)	0
Dried small-pool plasma with or without blood	867	0	1 (0.12%)	0
Dried irradiated large-pool plasma with or without blood	984	5	39 (3.96%)	1

A comparison of the results with kaolin-treated filtered liquid plasma and with whole blood showed that of 1,387 patients given whole blood, jaundice of doubtful cause developed in 3 and homologous serum jaundice attributed to the transfusion developed in 5 (0.36%); the comparable figures for the treated plasma were 1,366 patients, 8 of whom had jaundice of doubtful cause and 16 (1.17%), jaundice attributed to the transfusion. There were no deaths attributable to homologous serum jaundice in either series. It was concluded that the exposure of plasma to ultraviolet light under the conditions used did not inactivate the causative agent of homologous serum jaundice.

ITALY

Uncommon Findings in Hereditary Ataxia.—At the meeting of the Pistoia Medical Academy in December, 1953, Prof. Mattioli Foggia discussed three patients of the same family who had hereditary ataxia. Two other close relatives of the patients had died young with the same condition. This began gradually in all the patients with a staggering gait, a decrease of energy, physical decay, and a speech defect. The patients were a mother, her son, and her daughter. The condition had appeared in them at the ages of 60, 30, and 19 years, respectively. Some remissions had occurred in all three, nystagmus and lesions of the pyramidal pathways were absent, the sphincters were not involved, and all had a staggering gait without Romberg's sign. An intention tremor was present in the mother and the son but not in the daughter, and the mother had a congenital talipes equinus-like malformation of the foot and marked amyotrophy of the hands. Roentgenograms of all three patients showed no hypoplasia of the posterior cerebral fossa. Optic atrophy and syphilis or other intoxications were also absent. The differentiation from familial multiple sclerosis and the atypical forms of hereditary ataxia (Marie-Friedreich) and problems of neuropathology related to the genetic mechanism of these forms of hereditary ataxia were considered.

One of the problems was to establish whether in these patients the ataxia was associated with myotonia congenita or whether it was a polymorphic, pathological muscular manifestation that appeared with a myotonia congenita-like disease in the daughter and with a myasthenia-like disease and pronounced amyotrophy in the thenar and hypothenar eminences in the mother. The speaker could not find a report of any patient in whom myotonia congenita or myasthenia was associated with hereditary ataxia. Another problem was to determine whether in these patients the condition was primary or secondary. With a cathodal oscillograph an electromyographic test of the flexor and extensor muscles in the areas of myotonic disturbances seen in the daughter was made. The findings indicated a lesion in the flexor muscles, manifested by groups of potentials that were higher and less frequent than normal. In the periods of rest there were small action potentials due partly to reflex phenomena and partly to local muscular activity. In general, the myogram was that of a tetanus due to motor units acting in a nonsynchronized manner with some episodes of fasciculation.

In the extensor muscles, the activity of the single units was even more subdivided into partial episodes, and single potentials with elevated voltage were observed. The slowness of the activity of the extensor muscles during the distension of the fingers with thrusts of long-lasting action potentials was characteristic. In some instances discharges were observed after relaxation, as felt by the patient. Considering the fact that primary impulses capable of regulating the normal behavior of the innervation of the antagonistic muscle groups derive mostly from spindles and tendinous receptors, it may be that, in the presence of a lesion of the receptors in a muscle, the syndrome assumes the characteristics of cerebellar deficiencies, but the deficit represented by ataxia should in reality be considered secondary rather than primary. These three patients had a peripheral myopathy that exerted a secondary action on the spinocerebellar system and on that of the tonic mesencephalic nuclei.

Acute Erythropathy.—At a later meeting of the academy in February, Professor Di Guglielmo described a case of acute erythropathy due to chronic benzolism that presented alterations of the medullar erythropoietic system, some of which had never before been described. The patient was a printer in whom the condition had appeared suddenly; there was fever and progressive anemia, which led to death within one month. The diagnosis was based on the qualitative and quantitative alterations of the hemopoietic system. Sternal puncture revealed many cellular elements, mostly of the erythrocytic series, and this indicated a process of hyperactive erythropoiesis. Some of the erythroblasts were three or four times the normal size. Polyploid cells with two to eight nuclei and cells with the so-called secondary nuclei, nuclei divided into various lobes connected by bridges, or gigantic nuclei were seen.

These alterations were present not only in the more immature elements, as is usually the case, but also in the cells in which the hemoglobin was beginning to penetrate into the cytoplasm. These alterations suggested a blastomatous process; some authors speak of erythroblastoma when such lesions are present. Similar findings may be found in conditions that have nothing to do with tumors (such as pernicious anemia and hyperplastic processes of the myeloid tissue). They may also be found as an atypical hereditary trait in normal persons. These alterations are reversible in some patients. In this patient the findings described had disappeared almost entirely after 19 days. One day before his death the number of leukocytes increased from 6,000 to 280,000 per cubic millimeter. The starting point of these acute hemopathies is medullar aplasia. This is followed by the mobilization of extramedullary hemopoietic centers with the production of blood cells the maturation of which may be blocked. Centers of compensatory hyperplasia may be found in the bone marrow.

Intramedullary Fixation.—At a meeting of the Salento medical and surgical society in January in Brindisi, Professor Montemartini reviewed the various means of treating ununited fractures and said that crossed temporary intramedullar fixation is less dangerous than other techniques. He uses thin, inoxidizable metal wires, thus preventing those lesions of the marrow that are produced by thick nails. He concluded that the open treatment with temporary intramedullary fixation should not be used routinely in the treatment of ununited fractures and pseudoarthrosis but only as a supplementary technique. He advised continued observation so that the forces that might lead to a changed position of the fragments could be promptly neutralized. With this treatment, disturbances in callus formation and consolidation are reduced to minimum; damage to the medullary cavity is avoided; and fixation is controlled with roentgenograms.

Current Status of the Thymus.—At a meeting of the Tortona medical and surgical society in February, Professor Copello stated that little is known about lesions of the thymus. From the anatomic standpoint the thymus is a gland of internal secretion, since it is a lymphoepithelial formation with a connective tissue capsule, has a cortical substance with many leukocytes and a medullary substance with Hassal's corpuscles, but lacks

an excretory duct. From the functional standpoint, however, there is not as yet sufficient evidence to justify considering the thymus a gland of internal secretion. The physiological involution of the thymus immediately after the period of youth proves only that, if it does have an endocrine action, this is linked to the growth period. Experimental studies have shown that the growth of tadpoles favorably influenced by the presence of thymus extracts is due to the presence in such extracts of fats with a low molecular weight as well as to the concurrent presence of nutritious animal and vegetable substances. On the other hand, the absence of the gland or the abolition of its function in young persons has had no definite adverse effects on physical and mental development.

The genital organs and the adrenals exert a direct action on the volume of the thymus (if these do not function, the thymus becomes enlarged), but the thymus does not exert any direct action on the genital organs or the adrenals. Hyperfunction of the thyroid and the anterior lobe of the hypophysis causes hypertrophy of the thymus, and their hypofunction causes atrophy of the thymus, but the thymus cannot modify the function of these endocrine glands. In view of these facts and the fact that the thymus is not indispensable to life, it seems questionable to ascribe to it a precise function. Moreover, the anatomic modifications of the thymus associated with myasthenia gravis do not clarify the problem, since the cause of these alterations is obscure. They may be caused by the ionic balance, primary muscular alterations, or endocrine alterations in several glands.

It is certain only that the thymus has a lymphocytic function. As regards treatment, it is still debatable whether an enlarged thymus should be irradiated. This treatment may relieve some patients who present evidences of lymphatic diathesis.

Biliary Pathways.—At a meeting of the "Filippo Pacini" medical academy of Pistoia in February, Professor Torsoli and his associates discussed the modern cholangiocholecystographic tests used to study the biliary pathways. The new synthetic preparations for cholecystography have led to a marked decrease in the number of noninformative cholecystopathic findings. Those that can be given intravenously have made it possible to carry out cholangiographic studies in a patient whose gallbladder has been removed or is functionally excluded. Iodopanoic acid (Telepaque) is a tri-iodine and is given by mouth. When Biligrafin, which has a higher iodine content and is given intravenously, is used during the painful attacks, not only the calculi but also the dyskinetic movements that cause the pain can be seen. Often the calculi and the painful spasm were found not to be in a close topographical relationship.

The speakers stated that most syndromes involving biliary dysfunction may be studied radiologically not only because it is possible to visualize the excretory ducts but also because it is possible to inject the gallbladder in patients in whom previous cholecystographic findings have always been noninformative. Only with the new intravenous test is it possible to distinguish cholecystic from hepatic function. The investigation of the latter by means of the elimination of the contrast medium has given results that agree with those deduced from such tests as the Quick test and the Bufano test. The new contrast mediums may cause cloudy swelling and fatty degeneration of the liver and kidneys, but only when given in twice the regular dose. The damage, however, is reversible in the early stages and should not, therefore, contraindicate the use of these drugs.

Studies on the Liver Function.—At a meeting of the Pavia medical and surgical society in February, Professor Didone discussed the results of some studies on the function of the liver in patients with abdominal diseases requiring operation. In 75 such patients a cephalin-cholesterol flocculation test, thymol turbidity test, determination of total cholesterol, and other tests were done to evaluate the frequency and the nature of the liver dysfunctions before operation. In 10 patients with common duct stones the chief alterations were attributable to obstruction, and signs of hepatocellular damage were few. These were evident, however, in varying degree and intensity in five patients with neoplastic biliary obstruction. This is ascribed to the fact that in some patients the cancer does not lead to complete obstruction of the extrahepatic biliary pathways. Patients with lesions of the gastrointestinal tract presented a high rate of hepatic dysfunction; 12 patients with gastroduodenal ulcer had signs of slight hepatocellular alteration. This was even severer in 12 patients with neoplasm of the stomach, 9 with neoplasm of the rectum, and 3 with neoplasm of the colon. Metastases to the liver secondary to gastrointestinal neoplasm in 10 other patients did not cause true alterations of the hepatic function. Professor Didone emphasized the importance of the functional investigation of the liver in patients with abdominal diseases requiring operation as well as in patients with diseases of the biliary pathways.

Renal Biopsy.—At a meeting of the Rome Medical Academy in February, Professor Malizia and his associates reported on the indications and usefulness of renal biopsy in the diagnosis of intercapillary nodular nephrosclerosis. The technique used by them consists in the visualization of the kidney by means of retropneumoperitoneum and the removal of a specimen with Sylverman's needle under visual control. In one patient they made a diagnosis of diabetic nephrosis despite the fact that the clinical findings and course were not typical of this condition. Renal biopsy is used not only in the diagnosis of diabetic nephrosis but also to study the course of the disease and to guide its treatment. It should be used, however, only on patients whose condition cannot be diagnosed clinically.

Jaundice in Patient Taking Phenylbutazone.—At the same meeting Professor Lucherini and his co-workers reported on a case of jaundice that developed during a course of phenylbutazone therapy. This is the 10th such case reported in the world literature. The patient, who had a severe form of chronic rheumatism, was receiving phenylbutazone in regular doses. Shortly after the treatment was started, gastric disturbances appeared and were followed by jaundice, subfebrile temperature, and pains in the right hypochondrium. Because this was believed to be a toxic jaundice caused by the phenylbutazone, the administration of the drug was stopped, but when the jaundice continued for a length of time longer than is usual in jaundice caused by drugs, a liver specimen was obtained for biopsy. This revealed histological alterations characteristic of viral hepatitis. When the signs of liver impairment had disappeared, the administration of phenylbutazone was resumed. This resulted in a marked improvement in the joints and no return of the jaundice. From this evidence it does not seem likely that the jaundice was caused by the drug.

Pseudohemicranial Headaches.—At a meeting of the medical society of Leghorn, Professor Greppi of Florence distinguished the true essential hemicrania of youth from the analogous syndromes that are caused by vascular lesions and dysfunctions. The latter are commoner in presenile and senile patients. The so-called pseudohemicranial headaches belong in a third group. These appear in the presence of inflammatory or productive processes in the endocranial and extracranial arteries. An arteritis or thromboangiitis localized in the superficial temporal artery and in other branches of the external carotid may be seen in old patients in whom atheromatous degeneration or arteriosclerosis with or without hypertension is present. Analogous findings may be caused by arteriosclerosis in elderly persons in whom the clinical characteristics of histamine cephalalgia appear even without the inflammatory or thromboangiitic aspect of the process.

Exfoliative Cytology in Gynecologic Conditions.—Professor Laudadio reported on exfoliative cytology at a meeting of the Tortona medical and surgical society. He said that the histiocytes found in smears from the female genital tract may easily be mistaken for neoplastic cells. Despite false positive and false negative reports, the method is of great value, especially if it is used as a supplement to other methods such as biopsy. The cytological examination has its greatest value in the detection of cancer in the 0 phase (called intraepithelial or preinvasive) and in the 1 phase, which in its subgroup *a* includes the subclinical or asymptomatic cases. At the meeting of the Foggia medical and surgical society in February, Professor Mele said that exfoliative cytology as a means of early diagnosis of malignant tumors of the female genital tract has become the commonest diagnostic method because of its simple technique and its certain results.

CORRESPONDENCE

RADIOLOGICAL TECHNICIAN

To the Editor:—X-ray technicians on the west coast of the United States were recently circularized by an organization offering memberships that authorized placing "R.T." after the name of the technician. This was to signify "Radiological Technician." These memberships were to be granted merely on application and the payment of a fee. There was nothing in the structure of the organization that would guarantee any particular competency in radiological technique on the part of the members. The consummation of this scheme would have, of course, confused the issue completely in the field of radiological technique in that, as is explained below, "R.T." has attained a well-defined meaning within the medical and hospital world. Fortunately, due to an inadequate response, the organization that considered entering into the program abandoned its plan. The American College of Radiology believes, however, that for the record and in support of the American Registry of X-Ray Technicians, which has since its inception operated under medical guidance, the dissemination of the following statement is desirable.

For the information and guidance of the medical profession in the United States, X-ray technicians, hospital personnel, and other interested persons, the American College of Radiology offers the following statement:

The American Registry of X-Ray Technicians, the headquarters of which is in Minneapolis, and whose executive secretary is Mr. Alfred B. Greene, R.T., is the sole organization offering certification or registration of X-ray technicians recognized by the American College of Radiology and the American Medical Association. The American Registry of X-Ray Technicians offers an opportunity for examination after the technician has followed a course of training approved by the American Medical Association, the American College of Radiology, and the American Society of X-Ray Technicians. These examinations are conducted by members of the American College of Radiology or trustees of the American Registry of X-Ray Technicians. Upon successful completion of examination the technician is registered and is authorized to place after his or her name the designation R.T. (Registered Technician).

It is the considered opinion of the American College of Radiology that the designation R.T. has, in the eyes of the medical profession, X-ray technicians, hospital personnel, and the public, acquired a well-defined meaning signifying: the successful undertaking of a given course of training, a specific level of achievement in X-ray technique, adherence to a well defined code of conduct, and passage of a supervised examination. The College would lament and discourage the action of any organization or person offering registration of X-ray technicians merely upon application rather than examination; beclouding the meaning of "R.T.," Registered Technician, by attributing to the initials some other meaning such as "radiological technician"; and misleading those concerned with competence in X-ray technique by sponsoring registration outside the purview of recognized medical and X-ray technician organizations.

WILLIAM C. STRONACH
Executive Secretary
American College of Radiology
20 N. Wacker Dr., Chicago 6.

BALLISTOCARDIOGRAPHY

To the Editor:—In their article, "Use of Ballistocardiography in Evaluation of Paroxysmal Rapid Heart Action," in THE JOURNAL, July 3, 1954, page 897, Drs. Selinger and Levin state in the discussion following case 1: "It is known that paroxysmal auricular tachycardia may be the first sign of coronary artery disease. If attacks of paroxysmal auricular tachycardia develop in a man past 40, one must suspect coronary disease as the cause." I do not believe that this represents the opinion of most cardiologists. The authors' opening paragraph expresses rather, I believe, the consensus; that is, that tachycardia "is fundamentally a functional disturbance, but one occurring *in* [italics mine] hearts that are structurally either normal or abnormal." Paroxysmal auricular tachycardia is found far more often in normal than in damaged hearts. Even in the middle-aged the finding of paroxysmal auricular tachycardia tends to reassure me that I am more likely to find the heart organically sound. One does not suspect coronary artery disease but looks rather for anxiety, tension, or fatigue as the probable cause. Prinzmetal, whom the authors cite, says paroxysmal auricular tachycardia "is essentially functional in origin. When it occurs in diseased hearts there is no constant association with any specific lesion." Although it may appear as the first sign of coronary artery disease, it is apparently coincidental (The Auricular Arrhythmias, Springfield, Ill., Charles C Thomas, Publisher, 1952, p. 46).

At the Los Angeles County General Hospital in 1945 the arrhythmias found in 1,247 electrocardiographic records of patients with acute myocardial infarction were reviewed. Paroxysmal auricular tachycardia was found only five times—surely no more than a chance relationship. It is doubtful that the relationship is any different in coronary artery disease without infarction. (See my article in *Am. Heart J.* 37:425 [March] 1949.) On the evidence, therefore, paroxysmal auricular tachycardia appearing in a middle-aged patient should not lead one to suspect coronary artery disease as the cause. If they coexist, it is coincidental.

JOHN MARTIN ASKEY, M.D.
1930 Wilshire Blvd., Suite 902
Los Angeles 5.

FUNCTIONS OF A MORTALITY COMMITTEE IN A COMMUNITY HOSPITAL

To the Editor:—One of the most difficult requirements of the Joint Commission on Accreditation of Hospitals is that the hospital staff shall discuss deaths. Such discussions are especially hard to conduct in the community hospital, where individual physicians are, naturally, reluctant to have the cases treated by them discussed before an open staff meeting.

To fulfill this requirement fairly and reasonably, the mortality committee is composed of representatives from the medical, surgical, obstetric, and pathological departments. Each death during the month is listed with physician, autopsy findings, date of admission, and date of death. This information is compiled before the staff meeting and handed to physicians at the meeting. Any staff member is welcome and encouraged to raise any discussion concerning these deaths. Obviously, it is impossible and impractical to discuss each death in detail, so deaths are placed in two categories.

For category 1, the mortality committee may each month select from the list of all deaths one or two that should be discussed because of their special interest or teaching value. The attending physician is asked to prepare a summary that is also handed to physicians at the time of the staff meeting. The attending physician then reads this summary at the staff meeting, and the pertinent points in diagnosis and treatment are discussed. Should a physician fail to prepare such a summary or fail to present it, a member of the mortality committee prepares and reports the case.

Category 2 consists of all deaths that occur following operation. Thus, a summary is automatically required for any patient who was operated on and who died during that admission. The operating surgeon presents the summary. Again, if the physician fails to write the summary, it will be prepared and reported by the mortality committee.

A mimeographed paper listing the categories is prepared each month and presented to each physician at the staff meeting. The chairman of the mortality committee leads the critique and encourages any discussion on questions regarding the deaths, which are usually not of sufficient interest to warrant lengthy discussion. In the category 1 type of deaths, which are of particular interest, various physicians are asked to discuss and criticize, or commend, the management. The same procedure applies to the deaths after operation in category 2. In this manner, all deaths are at least presented before the staff. Furthermore, the method is a useful one by which the community hospital may carry out, in part, the requirement of the Joint Commission on Accreditation that a thorough review and analysis of clinical work done in the hospital—on at least a monthly basis—be reported in writing.

ROBERT A. BUYERS, M.D.
Montgomery Hospital, Norristown, Pa.

GOVERNMENT SERVICES

AIR FORCE

Five Year Training Program.—The nine carefully selected regular Air Force medical officers who completed the advanced course at the School of Aviation Medicine, Randolph Field, Texas, Aug. 19, have embarked on a training program that covers five years and will qualify them as specialists in aviation medicine. The nine graduates had previously completed the primary course for squadron surgeons given at the school. The year of study just completed is the first of two years, the second of which will be taken at a civilian medical school. Two men are going to Johns Hopkins University and the other seven to Harvard. After finishing the two years of academic training and a two year residency at Air Force medical installations the officers will be eligible to take the board examinations in aviation medicine. In the group of officers taking this special training, which will be given to one class of about the same size each year, the Air Force expects to find its future leaders in research, education, and administration. The nine graduates were Majors George R. Anderson, David H. Beyer, Richard M. Fenno, John P. McCann, Robert K. Quinnell, and Raymond A. Yerg, and Captains Henry C. Moritz Jr., Walter H. Smartt, and Charles W. Westerbeck.

NAVY

Personal.—Vice-Admiral Clarence J. Brown, M.C., retired, has been appointed medical consultant to the department of the Youth Authority, state of California, with headquarters in Sacramento. Until his retirement on May 1, Admiral Brown served as Deputy Surgeon General of the Navy.

PUBLIC HEALTH SERVICE

Grants for Medical Research.—The Surgeon General has approved federal grants totaling $14,685,671 for 1,442 medical research projects in the major diseases afflicting Americans. The grants were approved during recent meetings of the seven national advisory councils. Four hundred fifty-nine of the awards, totaling $4,568,073, were for new research projects, and 983, totaling $10,117,598, were for continuation of existing projects. The awards were made to scientists in 335 research institutions in the United States and are administered by the National Institutes of Health, research bureau of the Public Health Service. These amounts represent about 60% of the fiscal year 1955 funds appropriated by Congress for medical research projects ($33,918,000) supported in whole or in part by Public Health Service grants. The grants award program covers support of research in the medical and biological sciences, particularly research in the causes and treatment of heart disease, cancer, mental illness, arthritis and other metabolic diseases, the neurosensory and neuromuscular diseases (glaucoma, retrolental fibroplasia, multiple sclerosis, cerebral palsy, and epilepsy) diseases of the teeth and oral cavity and certain diseases of microbiological origin, such as influenza, infectious hepatitis, the common cold, and malaria. Other projects supported by PHS research grants are concerned with fundamental exploration of metabolic and biological phenomena underlying the causes of most of the noninfectious diseases.

The Public Health Service is assisted in its evaluation of research grant applications from medical scientists by two types of advisory and consultative bodies. The first of these, called "study sections," consists of 17 panels of more than 200 leading technical experts, representing many branches of modern medical science. The second group consists of the seven national advisory councils established by Congress and composed of leaders in the medical sciences, in education, and in public affairs.

Before applications for research grants can be approved by the Surgeon General, they must first be approved by an advisory council. Each of the councils reviews the actions recommended by the study sections, supports or changes the recommendations, and sends the applications to the Surgeon General.

Institute of Arthritis and Metabolic Diseases.—Dr. DeWitt Stetten Jr. has been appointed associate director in charge of research of the National Institute of Arthritis and Metabolic Diseases, Bethesda, Md. The appointment was announced by Dr. Floyd S. Daft, director of the institute. Dr. Stetten will direct the institute's integrated program of fundamental research and clinical investigation into problems related to the several forms of arthritis and such metabolic diseases as diabetes, vitamin deficiencies, gout, obesity, and disorders of the blood, bones, and liver. He was, for several years, chief, division of nutrition and physiology, Public Health Research Institute of the City of New York, Inc. He has served also as assistant professor of biological chemistry at Harvard medical school, Boston, and assistant professor of biochemistry, College of Physicians and Surgeons, Columbia University, N. Y. He is a member of the editorial boards of four medical journals, the *American Journal of Medicine, Metabolism, Journal of Chronic Diseases,* and the *Journal of Biological Chemistry*. He received his undergraduate degree from Harvard College, and his M.D. and Ph.D. from Columbia University.

Medical Aid for Flood-Stricken Pakistan.—President Eisenhower dispatched by air last week end an 86-man emergency medical force to assist flood-stricken East Pakistan. Dr. Alexander D. Langmuir, who heads the force, took with him a half dozen of the Public Health Service's experts in fighting large-scale epidemics. Dr. Langmuir is the chief epidemiologist of the Public Health Service's Communicable Disease Center in Atlanta, Ga. Reports from Pakistan to the Foreign Operations Administration indicate that 7 million persons have been left homeless or seriously affected by the unprecedented floods that have followed the monsoons. Chief concern of the Pakistan government is the threat of major epidemics of typhoid fever, dysentery, cholera, and malaria. The Army has already dispatched to Pakistan 40 two-man teams of medical corpsmen to work with the PHS group.

Predoctorate Research Fellowships.—The Surgeon General announces a predoctorate research fellowship program at the National Institutes of Health in Bethesda, Md. These research fellowships in health, medical sciences, and related fields are available to successful candidates with bachelor's or master's degrees or equivalent training. Basic stipends are: first year, $1,400; intermediate year, $1,600; terminal year, $1,800. In addition to the basic stipend, $350 allowance is made for spouse and each dependent child. Basic tuition and certain travel allowances are paid. Application may be made at any time. Information may be obtained from Research Fellowships Branch, Division of Research Grants, National Institutes of Health, Bethesda 14, Md.

VETERANS ADMINISTRATION

Personal.—The following two appointments to the staff of the Department of Medicine and Surgery, Washington, D. C., have been announced: Dr. Linus A. Zink, formerly director of hospitals and clinics, has been promoted to deputy director for operations. Dr. Irvin J. Cohen, formerly manager of the VA hospital at Baltimore, has been appointed to the central office post of deputy director for hospitals.

The Veteran Population.—The Veterans Administration reports that as of June 30, 1954, the total number of veterans in civil life was 20,850,000 and, of these, 15,425,000 were World War II veterans, 3,236,000 World War I veterans, and 2,189,000 veterans of other wars and the regular establishment. The last figure includes veterans with service only since the start of the Korean campaign and former regular establishment members of VA compensation rolls. On June 30 the total daily patient load in VA and non-VA hospitals was 109,640, of which number 4,790 patients were in non-VA hospitals. The total number of site acquisitions for new hospitals, as of June 30, 1954, was 66 and the total number of new hospitals completed was 60, while 6 new hospitals were still under construction. In the hospital addition and conversion program, 52 additions and conversions had been completed, 4 were in progress, and 2 other construction contracts had been awarded.

COMMITTEE ON MENTAL HEALTH

The following "Resolution on Relations of Medicine and Psychology" was developed jointly by special committees of the American Psychiatric Association, the American Psychoanalytic Association, and the Committee on Mental Health for the purpose of presenting a united front concerning the fact that "psychotherapy" is a special form of medical treatment that should be selected for use according to medical criteria, and that "psychotherapy" does not form the basis for a separate profession.

This resolution has been submitted to and approved for publication by the board of trustees of the American Medical Association, the council of the American Psychiatric Association, and the executive council of the American Psychoanalytic Association.

R. J. PLUNKETT, M.D., *Secretary.*

RESOLUTION ON RELATIONS OF MEDICINE AND PSYCHOLOGY

For centuries the Western world has placed on the medical profession responsibility for the diagnosis and treatment of illness. Medical practice acts have been designed to protect the public from unqualified practitioners and to define the special responsibilities assumed by those who practice the healing art, for much harm may be done by unqualified persons, however good their intentions may be. To do justice to the patient requires the capacity to make a diagnosis and to prescribe appropriate treatment. Diagnosis often requires the ability to compare and contrast various diseases and disorders that have similar symptoms but different causes. Diagnosis is a continuing process, for the character of the illness changes with its treatment or with the passage of time, and that treatment which is appropriate may change accordingly.

Recognized medical training today involves, as a minimum, graduation from an approved medical school and internship in a hospital. Most physicians today receive additional medical training, and specialization requires still further training.

Psychiatry is the medical specialty concerned with illness that has chiefly mental symptoms. The psychiatrist is also concerned with mental causes of physical illness, for we have come to recognize that physical symptoms may have mental causes just as mental symptoms may have physical causes. The psychiatrist, with or without consultation with other physicians, must select from the many different methods of treatment at his disposal those methods that he considers appropriate to the particular patient. His treatment may be medicinal or surgical, physical (as electroshock) or psychological. The systematic application of the methods of psychological medicine to the treatment of illness, particularly as these methods involve gaining an understanding of the emotional state of the patient and aiding him to understand himself, is called psychotherapy. This special form of medical treatment may be highly developed, but it remains simply one of the possible methods of treatment to be selected for use according to medical criteria for use when it is indicated. Psychotherapy is a form of medical treatment and does not form the basis for a separate profession.

Other professional groups such as psychologists, teachers, ministers, lawyers, social workers, and vocational counselors, of course, use psychological understanding in carrying out their professional functions. Members of these professional groups are not thereby practicing medicine. The application of psychological methods to the treatment of illness is a medical function. Any physician may utilize the skills of others in his professional work, but he remains responsible, legally and morally, for the diagnosis and for the treatment of his patient.

The medical profession fully endorses the appropriate utilization of the skills of psychologists, social workers, and other professional personnel in contributing roles in settings directly supervised by physicians. It further recognizes that these professions are entirely independent and autonomous when medical questions are not involved; but when members of these professions contribute to the diagnosis and treatment of illness, their professional contributions must be coordinated under medical responsibility.

WALTER B. MARTIN, M.D.
President
American Medical Association

ARTHUR P. NOYES, M.D.
President
American Psychiatric Association

IVES HENDRICK, M.D.
President
American Psychoanalytic Association

MEDICAL FILM REVIEWS

Fractures of the Humerus: 16 mm., color, sound, showing time 25 minutes. Produced in 1953 by Churchill-Wexler Productions, Los Angeles, for and procurable on loan from Presentation Division, the Veterans Administration, Vermont and H St., N. W., Washington 25, D. C.

The anatomy of the bones and muscles with their pulls that cause displacements is demonstrated. In each fracture the dangers of ill-advised manipulations are shown. Methods of reduction and maintenance of position are demonstrated, and the reasons for various manipulations are brought into bold relief. This film is highly recommended for medical students, interns, and residents. It will also serve as an excellent review for orthopedic surgeons. The photography and animation are exceptionally well done.

Operative Procedure for Total Urinary Incontinence (PMF 5202): 16 mm., color, sound, showing time 20 minutes. Produced in 1952 by the Department of the Army. Procurable on loan from Surgeon of the Army area in which the requestor resides.

This is a case-report type of film depicting on the cadaver the operative technique of using fascial transplants of the external oblique muscle that are swung down through tunnels under the symphysis and the membranous urethra. The technique demonstrated had been used on one patient in whom total urinary incontinence developed due to injury of the external sphincter during transurethral prostatic resection. The film concludes with a few postoperative sequences of the actual patient during the act of voiding. No justification was given for doing this elaborate procedure rather than simpler techniques used in the past. The surgical technique as shown was essentially correct. The photography is well done. This film will be of interest chiefly to urologists.

Functional Digestive Disease.—Functional disturbances of the digestive tract may be classified into 1) those caused entirely by a psychic factor such as emotional trauma or excessive nervous tension; they are transitory, easily identified and cured by relief of the instigating factor, 2) those having a psychic background, but which are no longer transitory but sustained in a groove of poor digestive function with habitual incoordination, and 3) those in which disturbance in coordination is caused by direct interference, through the use of food difficult of digestion, and excesses in smoking, alcohol and the use of laxatives. In the second and third groups the etiologic factors may be intermingled, to the extent that sustained central nervous disturbances may result in interference on the part of the patient by means of laxatives or roughage in food and, conversely, habitual interference may result in nervous disturbances. . . . Only in the first group with its transient manifestations of digestive dysfunction is the term psychosomatic exclusively applicable, and in these cases treatment for nervous disturbances alone is effective.—Sara M. Jordan, M.D., Functional Digestive Disease in the Aged, *The Journal of the American Geriatrics Society*, November, 1953.

THE LEISURE CORNER

FOR THE LOVE OF CHESS

Not too long ago as history goes, Dr. Sigmund Tarrasch, a German physician who had nearly attained the status of world champion chess player earlier in the century, summarized the satisfaction that is obtained from playing chess in a few, expressive words: "I have always had a slight feeling of pity," he declared in an essay on chess, "for the man who has no knowledge of chess, just as I would pity the man who remained ignorant of love. Chess, like love, like music, has the power to make man happy." That this experience is widely shared has been confirmed by the experiences of countless chess devotees over a period of some 1,500 years.

Although the precise origin of chess is submerged in a haze of obscurity, chess, as far as is known, dates back to India in the fifth century A. D. During the Middle Ages the game was introduced in Europe, where it soon became the most fashionable pastime of the leisured classes. (As a parenthetical reminder of the days when chess was regarded as both the sport of kings and the king of sports, it is interesting to note that chess is the only game permitted in the British House of Parliament.) Came the democratization of life, and the popularity of chess grew by leaps and bounds. By the middle of the 19th century chess centers were established in every large city of Europe, the British Isles, and the United States, and numerous magazine and newspaper columns devoted exclusively to chess attracted thousands of ardent readers. Today, chess is an integral part of our culture, distinguished by an endless stream of tournaments, club affairs, and literature dealing with every aspect of the game.

As a mere pastime chess is learned quickly, even though some persons regard it as weighted with sedation and far too difficult for anyone to master but mathematicians, statisticians, actuarial experts, and the like. Actually, a modest amount of study will enable many chess-disposed physicians to become pretty fair players. Like most games of skill, mastery of chess does take time and study; however, the beginner can extract considerable pleasure right from the start, and the pleasure thus derived is almost certain to multiply as the chess player's game improves. One obstacle to the spread of interest in chess has been its alleged difficulty. Even though chess is not as simple as tick-tack-toe, anyone can learn to play chess in half an hour. Naturally, there is more to chess than knowing how to move the pieces; however, the very complexity of the game is the source of its fascination. Indeed, one of the peculiar attractions of chess is that if two beginners are equally matched it is almost certain that they will feel the same interest in the game as though they were thoroughly versed in its intricacies.

Excellence in chess is attained only by persistent concentration on the game. A truly proficient player must not only learn the subtle variations in which the game abounds but also be able to apply his knowledge, resources, and powers of analysis in attack and defense. On the surface, chess provides a pleasant and refreshing diversion, but to some chess players the intensely competitive aspects of the game arouse deep-seated instincts. Dr. Emanuel Lasker, a mathematician who ruled the world of chess as a champion for many years, maintained that the spirit of combat during chess play was in itself rewarding and that two chess players competing over a board could aptly be compared to two generals on a battlefield, the strategy and tactics of chess being similar in spirit to actual warfare.

The argot of chess typifies the international flavor that has invaded languages the world over. The name of the game is derived from the Persian phrase "Shah mat," or "the king is dead." From the German stem such recognizable terms as "blitz" (lightning chess); "sitzfleisch" (the ability to patiently wait out an opponent); and "zugzwang" (forced move). From France come "en passant" (an uncommon form of capture of a pawn); "en pris" (attacked); and "j'adoube" (I adjust—relieving the player of the necessity of moving a piece that has been touched). Italian contributions include the words "fianchetto" (from the side); "gambit" (sacrifice, usually of a pawn); and "tempo" (time element). Yiddish has contributed the comical term "potzer," which means a clumsy or careless player.

The general type of chess pieces in use today was designed about a century ago in England. The names attached to chess pieces reflect the age of chivalry at the time of the Crusades, with the king, queen, bishop, knight, castle, and pawn characterizing the structure of medieval society. Chess is as rich in eponyms as is medicine. Openings in the game, traps, defenses, gambits, and positions have been named after famous chess masters, cities, and countries—for example, the Dutch opening, the Danish gambit, the Steinitz defense, and the Ruy Lopez opening.

Of the estimated eight million chess players in the United States alone, most devotees play chess across the board or by correspondence or else solve chess problems, which are artificial positions on the chess board permitting mate in a specified number of moves, with play involving elements of strategy or beauty. By chess players who are shut-ins or who live at great distances from each other, correspondence chess is played one or more moves at a time via the postal route. Thousands of correspondence players in the United States are grouped into correspondence clubs publishing their own magazines, conducting their own tournaments, and crowning their own champions. While it is not generally known, chess has also been played by cable, telegraph, radio, telephone, and semaphore.

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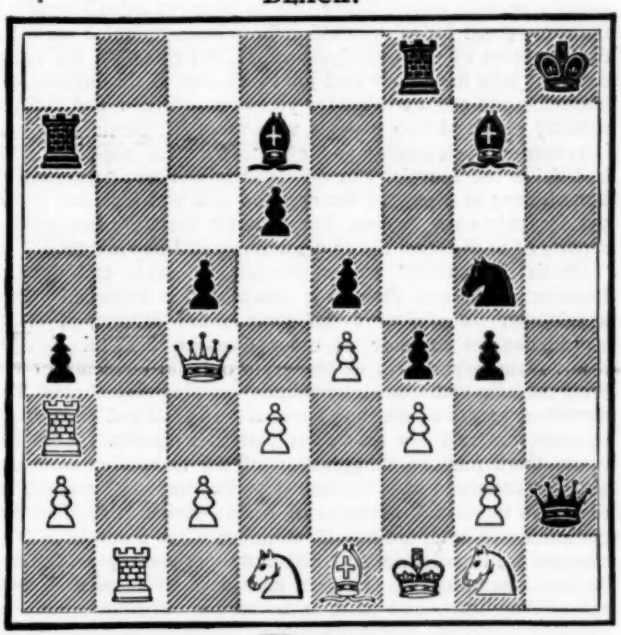

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Chess is an ideal form of recreation for physicians because while the game is played it is impossible to be concerned with the trials of medical practice, the national business picture, or the pros and cons of the international situation. Whether he likes it or not, the chess player must divorce himself from thoughts of everything else in order to concentrate on the chess pieces and the board. As with most hobbies, chess serves as a method for making new friends. Physicians who travel often find the game to be a strong bond linking them to other medical colleagues. Even when language serves as a barrier, communication between two persons can be established effectively over a chess board.

Finally, a brief comment about the disadvantages of a fascinating hobby; for one, conversation languishes during a chess game, and for another, chess has so strong an attraction for some players that the compulsion to play the game almost amounts to addiction. Chess can become a time-consuming obsession, it is true, but so can any other game if it is taken too seriously. For most physicians who take the game in a lighthearted spirit and who have learned to play for the sheer fun of it, chess is a never-failing source of intelligent amusement.

MEDICAL LITERATURE ABSTRACTS

INTERNAL MEDICINE

Sickle Cell Disease: An Analysis of Recent Advances. J. E. Moseley and J. B. Manly. J. Nat. M. A. **46**:177-181 (May) 1954 [New York].

Although previously the frequency of the sickling phenomenon had been estimated to be approximately 7% in the American Negro, improvements in techniques have shown that the frequency is between 9 and 10%. It is further estimated that hemolytic anemia develops in 1 to 2% of those who have the sickling trait. After commenting on the abnormalities of the hemoglobin molecule in the sickling phenomenon and on the inheritance of sickling, the authors deplore the fact that, despite the frequency of sickle cell disease among the American Negro population, cases are frequently overlooked or incorrectly diagnosed. Many patients with abdominal crises of sickle cell anemia or with cholelithiasis due to pigment calculi resulting from increased blood destruction have been operated on without suspicion of the underlying cause. Patients with the sickle cell trait who have bone and joint pain due to thrombosis and infarction are often treated for years for arthritis. The authors recently observed two patients with gross hematuria in whom intravenous pyelography and cystoscopy were negative. Both had sickle cell anemia. The diagnosis in one was suggested by bone lesions in a part of the skeleton that was included in the pyelographic x-ray studies. The urologic literature had several reports of such cases. Many cases diagnosed as "idiopathic" or "essential hematuria" are attributable to sickle cell disease. Because sickle cell disease is essentially a disturbed blood physiology, its clinical manifestations are diverse; it rivals syphilis as an imitator of other diseases. Skeletal changes result from hyperplasia of the erythroblastic elements of the bone marrow and from intraosseous vascular thromboses. The medullary spaces of the long bones are widened and the cortices become thin and the trabecular pattern irregular. In the flat bones there may be exaggeration of the trabecular markings and the vertebrae may become demineralized and cupped. In the skull, there may be widening of the diploic space between the inner and outer tables with thinning of the outer table. The changes are usually most marked in the parietal region. Several distinct hematological disorders may develop in persons with the sickling trait, and so it is no longer sufficient simply to speak of sickle cell anemia without attempting to establish the genetic factors or the forms of hemoglobin responsible for the disease.

Death from Cat Scratch Disease. S. Gräff. Monatsschr. Kinderh. **102**:232-237 (April) 1954 (In German) [Berlin, Germany].

Gräff reviews the present status of knowledge on cat scratch disease, pointing out that it is a lymphadenitis, which, depending on the site of invasion of the causal factor, presumably a virus, may become manifest as a cutaneoglandular, a tonsilloglandular, or an oculoglandular type. According to Mollaret, the diagnosis can be verified with the aid of a complement fixation test some weeks after the appearance of the glandular process. Although it has been generally accepted that the disease runs a favorable course, terminating in spontaneous cure, Gräff reports the case of a 30-month-old boy who died after an attack of cat scratch disease. Cat scratch disease was thought of on the basis of the clinical aspects and the results of postmortem examination. Inquiry revealed that the child had played with a cat (kissing, etc.) and that the cat had been sick with diarrhea and vomiting. The boy had the tonsilloglandular type of the disease, which within about 10 days led to a generalized toxic infection. Histological studies revealed the typical reaction of cat scratch disease particularly in the lymphatic tissues of Waldeyer's tonsillar ring and the regional lymph nodes. The author lists factors indicative of infection by the cat, but objects to the term cat scratch disease, pointing out that scratching by the cat is not necessary and that other forms of contact may transfer the disease. He suggests the term felinosis as preferable to cat scratch disease.

Atabrine in Treatment of Tapeworm. K. M. Paeckelmann. Deutsches med. J. **5**:244-245 (May 15) 1954 (In German) [Berlin, Germany.]

Paeckelmann says that the efficacy of quinacrine (Atabrine) against tapeworm was discovered in 1944 in South America in the course of a prophylactic campaign against malaria. He reviews the literature and reports his own observations on the use of quinacrine in tapeworm. Some reports were rather indefinite as to the number of patients treated, but emphasized the efficacy and good tolerance for this drug. In 347 of 488 patients with Taenia saginata or Taenia solium, who were treated with quinacrine, the tapeworm with head was expelled, and, in an additional 46 patients, a tapeworm was expelled, but the head could not be found. The administration of quinacrine in the treatment of tapeworm was as follows: The evening before the patient generally abstained from eating. On the next morning, while still fasting, he was given 0.8 to 1 gm. of quinacrine. Two hours later, 20 to 30 gm. of a saline cathartic was given, and the tapeworm was expelled within two to four hours. Occasionally quinacrine may cause vomiting, and this may interfere with the success of the treatment. Quinacrine can be administered by the duodenal tube. This mode of administration increases the chances of success. The author feels that quinacrine approaches the requirements of an ideal anthelmintic more nearly than do preparations customarily used. The administration of quinacrine in 1 gm. doses is not likely to cause toxic manifestations, if contraindications to its use are absent. The anthelmintic dose can be repeated after several days if this should prove necessary. Success may be expected in from 80 to 90% of cases.

Intractable Angina Pectoris: Treatment with Fractional Doses of Radioactive Iodine. J. B. Wolffe, A. D. Dale and E. I. Siegal. J. Am. Geri. Soc. **2**:288-292 (May) 1954 [Baltimore].

Wolffe and associates feel that the irreversibility and risk associated with thyroidectomy in patients with angina have made this approach too drastic. Thiouracil and other antithyroid drugs carry with them the danger of agranulocytosis. The advent of radioactive iodine (I^{131}) has made available a new, effective and easily controlled therapeutic agent. This report is based on observations on 32 patients who were treated with radioactive iodine. Twenty-four were euthyroid, five were hypothyroid, and three were hyperthyroid. Four of the 32 patients with intractable angina experienced complete remission of symptoms with one dose of I^{131} (average, 10 mc.). Ten patients had some relief after one dose of I^{131} and complete remission of symptoms after a second dose. The average total dose was 22 mc. These 14 patients showed signs of depressed thyroid activity; in fact, the degrees of relief from anginal seizures in many instances seemed to be in direct proportion to the decrease in thyroid activity obtained with the use of I^{131}. Twelve patients experienced some relief of symp-

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The place of publication of the periodicals appears in brackets preceding each abstract.

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toms following two courses of I^{131} but showed minimal evidence of depressed thyroid activity. The two doses averaged 16 mc. A third dose has been administered to this group but reevaluation has not yet taken place. Six patients had no alleviation of symptoms following two courses of therapy with an average total dose of 18 mc. The authors feel that a combination of prevailing forms of therapy with I^{131} is more effective than either type of treatment alone. The underlying cause or causes of the anginal attacks should be treated.

The Heart and Gall Bladder Disease. J. R. Beckwith, A. G. Hampton, D. T. Kernodle and others. Virginia M. Month. 81:211-214 (May) 1954 [Richmond, Va.].

Beckwith and associates emphasize the apparent close association between coronary disease and cholecystitis and review the evidence that impulses occurring in the biliary tract and other abdominal viscera may act as trigger mechanisms in the production of various cardiac arrhythmias and angina pectoris. The differential diagnosis between the two conditions is often difficult because of the fact that the pain impulses may be transmitted to the same or overlapping areas in the spinal cord. They present histories of several patients in whom cardiac abnormalities subsided after cholecystectomy. They describe a case of gallbladder disease resembling angina pectoris and one of myocardial infarction resembling gallbladder disease. A review of the records of 107 men and 100 women who had undergone cholecystectomy revealed that, of the men, 10 died postoperatively. None of the women died. There were 38 men with heart disease, of whom 3 died as a result of this, and three died as a result of noncardiac conditions. It appears that a patient with coronary disease, particularly a man, incurs a mortality risk of between 5% and 10% when undergoing cholecystectomy. It is difficult to determine in a given patient what effect a diseased gall bladder has on the heart, but, in many patients with symptoms of cholecystitis associated with those of coronary disease or of arrhythmia, the risk of cholecystectomy is justified because of the likelihood that the gallbladder is a trigger mechanism that potentiates the symptoms of coronary disease or of arrhythmia.

Endomyocardial Fibrosis. J. D. Ball, A. W. Williams and J. N. P. Davies. Lancet 1:1049-1054 (May 22) 1954 [London, England].

The authors, who work in Uganda, Africa, suggest that endomyocardial fibrosis is the cause of much of the heart disease of obscure cause seen there. They analyze a series of 20 patients whose disease was proved at necropsy. In this condition, large areas of firm white fibrous tissue are seen on the endocardial surface of the ventricles, commonly involving the structures of the auriculoventricular valves, particularly the posterior mitral cusp and chordae, with resulting mitral insufficiency. In some cases an obliterative effect is produced, reducing the capacity of the right ventricle. Parietal thrombus is common. In Africa persons suffering from endomyocardial fibrosis are generally of the lowest economic level. Of this series of 20 patients, 5 died within a few days of admission to the hospital, but most patients lingered for weeks or months in a waterlogged condition unhelped by treatment. In some cases, intercurrent infection contributed to death. The authors' longest period of observation was 20 months; this patient had a history of recurrent attacks of edema for two or three years previously. Another patient had had symptoms for seven years. These were the longest histories among the 20, and both patients had endomyocardial fibrosis of the obliterative type with gross fibrosis that appeared to be of long standing. Evidently some patients can survive the initial injury for many years, fibrosis proceeding to a point at which cardiac disability becomes stationary or only very slowly progressive, as in rheumatic heart disease. A few patients have been seen at necropsy with localized patches of superficial endomyocardial fibrosis at the apex of one of the ventricles. In them the cause of death was not attributable to this lesion, and there was no reason, from the clinical evidence available, to suspect any impairment of cardiac function during life. The response to treatment of heart failure by bed rest with digitalis, mercurial diuretics, and aspiration of fluid was disappointing. Nothing is known about the earlier stages of this disease, and its cause is also unknown.

Eosinophilic Granuloma of the Lung. W. F. Mazzitello. New England J. Med. 250:804-809 (May 13) 1954 [Boston].

Eosinophilic granuloma of the lung without associated skeletal or visceral lesions was first described by Farinacci and others in 1951. They observed these lesions in two enlisted men, both of whom had chronic cough, weight loss, exertional dyspnea, and chest discomfort. The diagnosis was made by thoracotomy and lung biopsy. The histological appearance of the excised nodules had features identical with that of eosinophilic granuloma of bone. Mazzitello presents two similar cases that likewise were verified by thoracotomy and lung biopsy. The similarity of these pulmonary lesions to the isolated skeletal lesions of eosinophilic granuloma is pointed out, and a similar chronic benign clinical course is expected. Mazzitello mentions the studies of various investigators, particularly a recent review by Lichtenstein, in which the confusion in terminology is deplored, and the new term "histiocytosis X" is suggested as an all-inclusive name, with eosinophilic granuloma as a localized form, and Letterer-Siwe's disease and Hand-Schüller-Christian's disease (lipid histiocytosis of the cholesterol type) as disseminated forms. The term "histiocytosis" refers to the essential type of histological cellular reaction occurring in these diseases, and "X" indicates the need for the identification of an etiological agent. The treatment of the pulmonary lesions is still undecided. Many more cases will have to be evaluated to determine accurately the effectiveness of corticotropin, cortisone, and x-ray therapy.

Bronchial Carcinoma—A Pandemic. J. Clemmesen. Danish M. Bull. 1:37-46 (April) 1954 (In English) [Copenhagen, Denmark].

Clemmesen feels that the greater part of the increase in frequency of cancer of the lung, which became evident during the first years of this century, may well have been apparent only and caused by the prolongation of the average length of life together with progress in diagnostic technique, but it should be realized that the latter cannot change the sex ratio for cancer of the lung. From autopsy records in Denmark it appears that the increase in bronchial carcinoma cannot have begun before 1926 and the rise is doubtful up to 1930. The author raises the question as to whether air pollution plays a part in the increase in bronchial cancer. The incidence of lung cancer in districts free of air pollution does not indicate that this cancer will be reduced by complete abatement of air pollution. Inquiries have revealed a higher number of heavy smokers among patients with bronchial carcinoma than among control persons. However, since there are nonsmokers with bronchial carcinoma, and a large number of smokers without the disease, the figures often fail to convince lay readers. The author feels that from reported studies it seems impossible to escape the conclusion that a mortal disease, which demands decades for its development, and probably as lengthy efforts for its prevention, is now increasing on a pandemic scale because of widespread addiction to smoking.

Use of Hexamethonium in Arterial Hypertension. D. A. Rytand. California Med. 80:375-376 (May) 1954 [San Francisco].

According to Rytand, hexamethonium properly given reduces blood pressure appreciably in most hypertensive patients. With the patient under careful observation in a hospital, the first two doses of the drug several hours apart should not exceed 5 mg. each, in order to avoid syncope or more severe depressor reactions. Since tolerance appears promptly, the dose must be increased during the next few days or even weeks according to the patient's response. This requires careful observation and many determinations of arterial pressure levels, especially in the hour or two immediately following an injection. The use of the drug is associated with prominent and unpleasant side-effects; fall of pressure sometimes precipitates acute myocardial infarction, hemiplegia, uremia, or even death. Hexamethonium

is, therefore, suitable for relatively few hypertensive patients (rather young ones with severe or malignant syndromes) and often fails in those with renal insufficiency who most need help. Of 24 hypertensive patients so treated, 12 died of the disease, and, of the other 12, only 2 cared to go on with the injections after one year; symptoms were too unpleasant and results too unimpressive for the others. The degree of care required to obtain satisfactory effectiveness is such that the program of treatment becomes too unwieldy for general use.

Acute Pancreatitis; Clinical Study of One Hundred Cases. W. F. Becker. J. Louisiana M. Soc. **106**:166-170 (May) 1954 [New Orleans].

One hundred consecutive cases of acute pancreatitis observed between 1943 and 1952 at the Charity Hospital in New Orleans are reviewed. Only those patients are included, in whom laparotomy or autopsy demonstrated acute pancreatitis or who presented a consistent clinical picture in conjunction with an increase in serum amylase. The number included 84 cases of acute edematous pancreatitis and 16 cases of acute pancreatic necrosis. Alcoholism has been described as a predisposing factor in acute pancreatitis. Twenty-two per cent of the patients in this series were chronic alcoholics, and in 13% the onset of acute abdominal pain was known to have followed ingestion of alcohol. Four patients had acute pancreatitis within three months after termination of pregnancy, and in each instance there had been similar but less severe attacks during pregnancy. The sudden onset of severe abdominal pain was the initial symptom in over 90% of the cases. In 13 cases it appeared shortly after the ingestion of a heavy meal. The pain was usually severe and steady and first involved the upper abdomen. Nausea was experienced by 90% of the patients, and vomiting occurred in 82%. A firm, fixed, tender abdominal mass was present in 11 cases. The mass was in every instance a pseudocyst or abscess and occurred only in the patients with acute pancreatic necrosis. The mortality of the series was 12%; of the 84 patients with acute edematous pancreatitis 3 died, of the 16 with acute pancreatic necrosis 9 died. One-third of the patients were subjected to early operation, in most instances because of errors in diagnosis. This poor diagnostic showing can in part be ascribed to the fact that during the early years of this study facilities for the performance of the serum amylase test were not available in this institution at night. A preoperative determination of the serum amylase was performed in only 18 of the 34 patients subjected to operation. Conservative treatment is best in most cases of acute pancreatitis. Early operation should perhaps be restricted to those instances in which the diagnosis cannot be established with reasonable certainty or when there exist signs of biliary tract obstruction, suppuration or cystic accumulations. Acute pancreatitis is now being recognized with increasing frequency largely because of a keener awareness of the condition and the more frequent use of the serum amylase test. At the Charity Hospital in New Orleans acute pancreatitis seems to be about one-tenth as common as acute mechanical intestinal obstruction and about one-fiftieth as common as acute appendicitis.

Salicylates and the Plasma Level of Adrenal Steroids. R. I. S. Bayliss and A. W. Steinbeck. Lancet **1**:1010-1011 (May 15) 1954 [London, England].

The possibility that salicylates act as antirheumatic agents by increasing the secretion of adrenocortical steroids either by stimulating the endogenous production of corticotropin or by directly stimulating the adrenal glands was investigated by measuring the plasma level of circulating adrenocortical steroids before and after salicylate therapy, since an increase in the secretion of the steroids by either of the mechanisms suggested would presumably lead to an increase in their plasma level. Acetylsalicylic acid was given to seven patients with either acute rheumatic fever or moderately severe rheumatoid arthritis at intervals of four hours in dosages ranging from 0.75 to 1.75 gm., and to four other patients in a single large dose of from 3.3 to 5.3 gm. in water. No significant increase in the level of the circulating adrenocortical steroids could be detected in either group of patients, although high plasma salicylate levels were obtained in three patients and were accompanied by salicylism severe enough to require reduction of the dosage in one. These findings indicate that the pituitary-adrenal system is not stimulated by salicylates given in clinical dosage.

Rheumatoid Arthritis: An Evaluation of Long-Term Treatment with Cortisone. E. P. Engleman, M. A. Krupp, W. W. Saunders and others. California Med. **80**:369-374 (May) 1954 [San Francisco].

Engleman and co-workers report on 36 men and 20 women with rheumatoid arthritis treated continuously with cortisone for periods ranging between 4 and 38 months. Fifteen patients received daily doses of 15 to 50 mg. of cortisone, 28 received 51 to 75 mg., and 13 received 76 to 100 mg. Concomitant therapy included periods of rest, physical therapy, and salicylates in daily doses of 2.4 to 3.6 gm. The incidence of subjective improvement exceeded that of objective improvement. Forty-nine patients (87.5%) had at least some decrease in pain and stiffness and significant increase in agility and joint mobility. The resulting functional improvement was usually sustained as long as cortisone was continued. Functional improvement was often observed despite obvious progression and increasing severity of the disease process as revealed by roentgenograms. Twenty-one of the 49 patients who were improved subjectively and functionally were classified on the basis of objective evidence as unimproved or worse. The incidence of objective improvement was higher in women; also, in those patients whose disease was in an early stage and of short duration at the time therapy was begun, and who required relatively smaller maintenance doses of cortisone. Therapeutic results were not affected by the age of the patient or by the presence of spondylitis. Despite precautions, the long-term administration of cortisone was, in some patients, productive of serious undesirable side-effects. Moonface was seen at some time during the administration of cortisone in all but four patients. Hypertension was observed in 13 patients, in 3 of whom it was uncontrolled despite reduction to the minimal effective dose of cortisone. Cerebrovascular accidents resulting in death occurred in two patients. The role of cortisone in those vascular accidents is moot. Duodenal ulcer occurred in two patients. Pathological fractures of vertebrae occurred in two women aged 77 and 52, both of whom had severe osteoporosis before the administration of cortisone. Final opinion regarding the role of cortisone in the treatment of rheumatoid arthritis must await the elapse of more years. At present it is obvious that cortisone is not a substitute for conservative and well-established measures. Combined with them, it provided in many cases a degree of social and economic rehabilitation, which occurred even in the presence of advancing disease.

Fatal Influenza. E. L. Walsh and F. Levy. West Virginia M. J. **50**:125-127 (May) 1954 [Charleston, W. Va.].

The case of fatal influenza presented by Walsh and Levy was that of a man, aged 27, who was admitted to the hospital with the complaint of cough of two weeks' duration. Five days before admission he awoke with chills, fever, and generalized aches. He received 200,000 units of penicillin daily for three days before admission. His sputum became rust colored two days before admission. He continued to have chills and fever each day, and his cough remained unchanged. There was shortness of breath and a sense of fullness. Roentgen examination of the chest on admission showed pneumonia in the lower lobe of the right lung. The heart was somewhat enlarged. Four days later, roentgenograms showed the pneumonia clearing but a pneumonitis beginning in the upper lobe of the right lung. Ten days later, the pneumonic process had cleared completely. On the day before death, a roentgenogram of the chest showed patchy pneumonitis in the upper and lower lobes of the right lung. At various times during hospitalization the patient was given digitalis, oxytetracycline (Terramycin), penicillin, streptomycin, plasma, cortisone, and corticotropin, but to no avail. Microscopic studies of necropsy material revealed large hemorrhages into the pectoral muscles and the overlying subcutaneous fat. There

was petechial bleeding into the pleura, diaphragm, pericardium, stomach, liver, gallbladder, and small intestines. Besides common bronchopneumonic changes, emphasis is placed on "desquamated epithelium" and especially the peculiar hyaline deposits in the alveoli. The authors cite other reports on fatal influenza, particularly Wolbach's of 1919, which emphasized that "death from influenza means death from lung complications." The hyaline deposits were also mentioned by other investigators. Opie emphasized in 1928 that "this hyaline material may form a continuous layer resting on the wall of the alveolar ducts and extending across the orifices of the tributary alveoli."

Diabetic Neuropathy: Controlled Clinical Trials. C. R. Shuman and S. F. Gilpin. Am. J. M. Sc. **227:**612-617 (June) 1954 [Philadelphia].

Three agents—vitamin B_{12}, pregnant mammalian liver extract, and adenosine triphosphate with thiamine or pantothenic acid—were given a therapeutic trial in patients with diabetic neuropathies that had not responded to careful regulation of diabetes with insulin and diet. In general, these were the severer neuropathies. Vitamin B_{12} was administered to 12 patients, with failure to observe improvement in their neurological disorder. Pregnant mammalian liver extract was equally ineffective in 15 patients. Subjective improvement occurred in three patients of this group, but it was found that two of these were receiving a placebo that was being used as a control in this phase of the study. Adenosine triphosphate with thiamine produced subjective improvement in two of six patients so treated. No effect was noted in those receiving pantothenic acid. There was no alteration in the objective manifestations of neuropathy with the use of any of the agents employed.

Prevention of Insulin Hypoglycemia. J. I. Goodman. Am. Pract. & Digest. Treat. **5:**464-467 (June) 1954 [Philadelphia].

The occurrence of hypoglycemic reactions has constituted a major problem in the management of diabetic patients. Since most reactions respond fairly promptly to glucose, administered orally or intravenously, there is a tendency to pass them off as harmless. However, when the brain tissue has been subjected to severe or prolonged hypoglycemia, widespread, bizarre, and often persistent clinical manifestations may be produced in spite of a normal or even an elevated blood sugar level. On the basis of case histories the author demonstrates that residual brain damage, transient hemiplegia and even death may result from insulin hypoglycemia. The protamine insulin group presents the greatest potential hazard insofar as hypoglycemia is concerned. This is true in the case of protamine zinc insulin, but especially so with mixtures of protamine zinc and regular insulin. The practice of insisting on the use of long-acting and intermediate insulins for every diabetic patient accounts for many reactions. In order to prevent hypoglycemia it is advisable to arrange the diet schedule to provide anticipatory feedings just before the time of maximum insulin action for each type of preparation, i. e., 2 hours after regular insulin, 6 to 7 hours after globin or isophane (NPH), and at bedtime (16 hours) in the case of protamine zinc insulin. Discussing the avoidance of insulin reactions during and after surgical treatment, Goodman says that he prescribes the identical dose of insulin that the patient has been taking right along. However, in patients who are unable to take food orally, 5 or 10% glucose solution is administered parenterally continuously until the patient is again able to resume normal eating. In the early stages of myocardial infarction there is a tendency to increase the dosage of insulin to compensate for the loss in carbohydrate tolerance. This practice may very well lead to hypoglycemia, sometimes with hazardous and even fatal results. Since the impaired sugar metabolism following a myocardial infarct is now believed to be part of the stress reaction, it is probably much safer to tolerate hyperglycemia and glycosuria for three or four days and leave the insulin dose as it is. Hypoglycemic reaction must be feared in patients with vascular disease, because of the disastrous complications that may accompany circulatory collapse, aside from fresh myocardial infarcts.

Tung Nut Poisoning. E. Balthrop, W. B. Gallagher, T. F. McDonald and S. Camariotes. J. Florida M. A. **40:**813-820 (May) 1954 [Jacksonville, Fla.].

The tung nut tree, Aleurites fordii, long has been grown in China as a source of tung oil. Its introduction into the United States and the rapid spread of its cultivation in the southeastern states has created a great new American industry. The mature tung nuts are crushed and their oil is expressed for use in the paint industry and allied fields. The whole ripe nut looks like a large walnut, and the unhulled seed is similar to a Brazil nut. The mature nut fresh from the tree has a sweetish taste like a chestnut, while the older, stored nut has a pleasant toasted almond flavor that is spoiled by the rancid after-taste that tung oil leaves in the mouth. Tung nuts are toxic. Cultivation of the tung tree for commercial and shade tree purposes is spreading. Because of these facts, especially the use of tung trees in urban areas where children have access to the nuts, tung nut poisoning is apt to become commoner. Tung nut poisoning is discussed and the literature reviewed. The primary toxic agent in tung nuts is a foreign protein, an albumin, causing gastrointestinal symptoms (nausea, vomiting, diarrhea) in mild cases, with shock and respiratory depression in severe cases. The treatment of tung nut poisoning is symptomatic, as it is for any food poisoning due to a foreign protein. The administration of magnesium sulfate by mouth seems to denature the protein. Whether this change is accomplished by its cathartic action, by osmotic dilution of the offending protein, or by salting out of the protein by the sulfate ion is not known. Concomitant with the use of magnesium sulfate, fluid and electrolyte replacement therapy is indicated especially for severely poisoned patients suffering from anaphylactoid reactions and potassium deficiency. Central nervous system stimulants and oxygen should also be used when indicated. In an extreme case corticotropin might be administered intravenously.

SURGERY

Indications for Surgical Treatment in the Anemic Forms of Diaphragmatic Hernia of the Stomach. P. Santy, J. Favre-Gilly and L. Laroyenne. Lyon chir. **49:**285-296 (April) 1954 (In French) [Paris, France].

The authors observed 16 cases of severe anemia produced by gastric hernia at the esophageal hiatus. Their experience has been that anemia occurs in about 25% of cases of diaphragmatic hernia. The syndrome is four times as common in women as in men. It appears in two different age groups—in young children and in adults between 50 and 60 years of age. Sometimes anemia follows hematemesis or melena; in other cases, the anemic symptoms are mixed with digestive symptoms, such as postprandial epigastric pain, or with pulmonary symptoms. The patients are often classed as having cardiac or coronary disease because of dyspnea or sternal constriction. Sometimes venous thrombosis is the presenting symptom, but often anemia is the only symptom and is classified as essential. Blood examinations usually show very hypochromic and microcytic but moderate anemia with a low mean corpuscular volume and corpuscular diameter. Chemical examination of the gastric juice is usually negative, but the chemical search for occult blood in the stools is usually positive (Weber reaction). Thus, this anemia appears to be posthemorrhagic. Radiological investigation is usually necessary to discover the hiatus hernia, either in simple radioscopic investigation or on series of gastric films and sometimes only in the Trendelenburg position. If the hernia is discovered first, as it was in one-fourth of the authors' cases, the possibility of anemia must not be neglected. If the anemia is known first, as it was in three-fourths of the cases, the possibility of Biermer, macrocytic, and refractory anemias must be eliminated. If microcytic and hypochromic anemia is found, a latent diaphragmatic hernia of the stomach should be suspected and radiological examination requested. Medical treatment consists of iron given orally or intravenously, but it only corrects the anemia and does not attack the cause. Surgery of the hernia is the only definitive treatment. Iron is used in the preoperative

period and anticoagulants in the postoperative period because of the high incidence of venous thrombosis and pulmonary embolism in this syndrome.

Vagotomy in Patients with Duodenal Ulcer. R. Jelinek. Wien. klin. Wchnschr. **66**:329-331 (May 14) 1954 (In German) [Vienna, Austria].

Of 136 patients who underwent vagotomy for nonbleeding duodenal ulcer between 1947 and 1951, one died of circulatory insufficiency caused by bronchopneumonia on the fourth postoperative day (an operative mortality rate of 0.73%). Of postoperative complications that occurred in only 14 patients, early vagus diarrhea in 7 patients was the most frequent. In 129 of the 136 patients, follow-up examinations were performed one to six years after the operation. Of the 129 patients, 77 had a vagotomy without gastrectomy, while 52 underwent combined vagotomy and gastrectomy. Of the 77 patients, 42 (54.5%) had no complaints whatsoever (highly satisfactory results), 17 (22.07%) had occasional general complaints that did not affect the subjective well being (satisfactory results), and 8 (10.38%) had complaints without roentgenologic evidence of ulcer (fair results). In 10 (12.90%) therapy failed. Of the 52 patients, 36 (69.23%) had highly satisfactory results, 10 (19.23%) satisfactory results, and 4 (7.69%) fair results. In two (3.48%) therapy failed. Thus results in patients who underwent combined vagotomy and gastrectomy were better than in those who submitted to vagotomy alone. Despite the relatively short follow-up of from one to a maximum of six years, the authors consider vagotomy combined with gastrectomy a valuable procedure in the treatment of duodenal ulcer.

Role of Surgery in the Therapy of Ulcerative Colitis. J. H. Garlock and A. S. Lyons. Gastroenterology **26**:709-722 (May) 1954 [Baltimore].

Of 254 patients with ulcerative colitis treated by surgical measures between 1937 and 1952, 145 had universal colitis with involvement of the entire colon and rectum, 97 had right-sided colitis in which the rectum and often the sigmoid were free of disease, and 12 had left-sided colitis sparing the right and transverse portions of the colon. The authors' concept of rational approach to the surgical treatment of ulcerative colitis was based on four premises: 1. Treat the pathological complications of the disease, namely, pericolonic abscess, fistula, hemorrhage, and perforation. 2. Save life in the occasional acutely toxemic patient who does not respond to conservative therapy. 3. Remove the colon after study indicates that irreversible pathological changes have taken place. 4. The development of polyposis makes colectomy obligatory because of the high incidence of carcinomatous change. Under such circumstances, surgical treatment encompasses complete rehabilitation from chronic invalidism to a healthy and useful life. In order to obtain a clear-cut picture of the value of surgical therapy for ulcerative colitis, all deaths from all causes, late or early, during or after hospitalization were included in tabulating mortality statistics. The case mortality as opposed to the operative mortality was stressed in order to obtain the exact percentage probability of survival of the individual patient. Of the 145 patients operated on for universal colitis, 26 died of all causes (a case mortality of 17.9%); 244 operations, including 125 ileostomies, 5 combined ileostomies and colectomies, 87 colectomies, and 27 abdominoperineal resections, were performed on these 145 patients, 18 of whom died of the operation (an operative mortality of 7.3%). Of the 97 patients operated on for right-sided colitis, 16 died of all causes (a case mortality of 16.4%); 195 operations, including 90 anastomoses, 3 anastomoses and colectomies in one stage, 79 colectomies, and 23 secondary ileostomies, were performed on the 97 patients, 16 of whom died of the operation (an operative mortality rate of 8.2%). Of the 12 patients operated on for left-sided colitis, 3 died of all causes (a case mortality of 25%); 23 operations, including 11 transverse colostomies, 11 colectomies and abdominoperineal resections, and one combined proximal colectomy and ileostomy, were performed on the 12 patients, 3 of whom died of the operation (an operative mortality of 13%). Thus of the total (254 patients), 45 died of all causes, a case mortality of 17.7%; 462 operations were performed on these 254 patients, 37 (7.9%) of whom died of the operation. Ulcerative colitis is probably the most difficult disease of the gastrointestinal tract to treat, but the authors hope that their report will help to bring some degree of rationalization to the two extremes of early radical surgical excision and denial of surgical intervention in any case.

Late Results in Treatment of 100 Cases of Acute Hematogenous Osteomyelitis. J. Trueta and J. D. Morgan. Brit. J. Surg. **41**:449-457 (March) 1954 [Bristol, England].

Trueta and Morgan state that while at the onset of the penicillin era patients with acute hematogenous osteomyelitis were treated with penicillin only, Trueta and Agerholm in a report published in 1946 gave reasons for the reintroduction of early surgery in the treatment of acute hematogenous osteomyelitis, using it as a complement to penicillin. This change was made when it was found that penicillin alone could not prevent the infection from becoming chronic. This report analyzes the results of treatments by penicillin and surgery in 100 cases of acute hematogenous osteomyelitis. Cases have been followed up for from two to eight years. The following treatment was used. On admission the patient was carefully examined by an experienced surgeon, and an estimate of the site and intensity of bony tenderness was made. Blood was then removed for culture, determination of the sedimentation rate, and white blood cell count. The affected part was immobilized and, if possible, elevated. Penicillin treatment was begun with a dosage of 1,000,000 units per day, given either by continuous intramuscular drip or by repeated injections at three hour intervals. Twenty-four hours after admission, the same surgeon reexamined the patient, observing the amount of tenderness of the suspected area, the temperature as shown on the four hour chart, and the sedimentation rate. Unless the tenderness had greatly decreased from that of the previous examination, the patient was prepared for operation. The skin incision is made over the area of maximum tenderness, the periosteum is split, and the subperiosteal abscess is evacuated; several small perforations are made through the cortex with a drill to relieve tension. The tourniquet is then removed, hemostasis is effected, penicillin powder, 200,000 units, is sprinkled into the wound, and the skin is sutured without drainage. The limb is then immobilized. Five days after the operation, the wound is reexamined and any hematoma is aspirated. The stitches are removed on the 10th day, and a plaster cast is applied. If the operation has succeeded in preventing involucrum formation, the bone will be particularly liable to fracture in the repair stage. Penicillin treatment is continued for three weeks. Immobilization is maintained for as long as signs of bone inflammation persist; after this the patient is allowed to walk or to use the limb according to the changes observed in the radiographs. Complete bone healing has so far been obtained in 66 cases. The quality of the results depends on the nature of the treatment and also on the age of the patient at the onset of infection. Complete healing was more frequent when the patient was closer to the beginning than to the end of childhood. Infants and adults present their own characteristic forms of the disease. Prolonged follow-up with radiological control is essential.

"Essential" and "Nonessential" Amino Acids in Urine of Severely Burned Patients. G. L. Nardi. J. Clin. Invest. **33**:847-854 (June) 1954 [New York].

The negative nitrogen balance of thermal trauma is recognized, but its meaning and significance to body metabolism are poorly understood. Partition of urinary nitrogen may assist in interpreting the significance of nitrogen loss. Some investigators have found an increase in nonprotein urinary nitrogen of burned patients. This was chiefly due to undetermined nitrogen, the latter defined as the difference between the total nonprotein nitrogen and the sum of urea, uric acid, creatinine, and alpha-amino nitrogen. To analyze this partition further, Nardi studied the urinary amino acid excretion in five healthy subjects (laboratory personnel), in six severely burned patients, and in patients undergoing surgical operations. He found an abnormal

amino-aciduria consisting of excretion of "essential" amino acids not normally present in urine as well as increased quantities of "nonessential" amino acids in the urines of severely burned or traumatized patients. The qualitative and quantitative aspects of this amino-aciduria may be proportional to the severity of the trauma. The amino acid excretion was influenced by, but not completely dependent on, the total urinary nitrogen. Hyperactivity of the adrenal cortex as seen in Cushing's disease resulted in a moderate degree of amino-aciduria. The latter effect was not reproduced by a daily maintenance dose of 30 mg. of cortisone after bilateral subtotal adrenalectomy. This escape of amino acids is most likely due to a failure of tubular reabsorption. The reason for the latter is not clear, but it may be due to some adrenal steroid other than cortisone, to an abnormal amino acid pattern presented to the tubule, or to some other factor.

Mediastinal Parathyroid Adenoma. C. E. Catlow. West. J. Surg. **62:**352-353 (June) 1954 [Portland, Ore.].

In the 50-year-old man whose case is reported, the history and the blood calcium values indicated hyperparathyroidism. Surgical exploration of the neck disclosed no parathyroid adenoma. It was decided that in a second operation the superior mediastinum should be explored. However, on the fourth postoperative day basilar pneumonia developed; it was followed by cardiac decompensation and death on the fifth postoperative day. Autopsy revealed a mass in the superior mediastinum, which on microscopic study showed the typical picture of a parathyroid adenoma. This case emphasizes the necessity of exploring the mediastinum after fruitless neck exploration.

A New Operation for Mitral Regurgitation. J. Hayward. Australian & New Zealand J. Surg. **23:**257-267 (May) 1954 [Melbourne, Australia].

Hayward suggests that most cases of mitral disease can be provisionally diagnosed as mitral stenosis or mitral regurgitation. Fused commissures are characteristic of typical mitral stenosis. These cases are ideal for valvotomy. Open or partly open commissures are typical of the valve of mitral regurgitation. This second group he subdivides into group 2a, in which the function of the posterior cusp is permanently destroyed, and group 2b, in which the function of both cusps is permanently destroyed. In addition to the two main groups, the author recognizes a small intermediate group with conical or elastic valve, in which stenosis and regurgitation may simultaneously be significant causes of circulatory trouble. In this intermediate group valvotomy is likely to be disappointing. Before describing his new operation for mitral regurgitation Hayward says that strips of pericardium, plastic substances, and other devices are being tried to relieve mitral regurgitation by attempting to produce either a solid buttress over the edge of or just under the posterior cusp, or a movable structure with valvular action below the orifice. None has so far proved satisfactory. In his new operation the left auricular appendage is invaginated through the mitral valve orifice and attached to the left ventricular wall under the posterior cusp. It is suitable for cases belonging to group 2a. The author presents the histories of two patients in whom this operation was employed, and he also lists advantages and limitations. In an addendum to this paper he admits that both patients after immediate encouraging results deteriorated to a condition similar to, or perhaps slightly worse than, their preoperative state. In each case the retrogression began gradually about six weeks after the operation. The reason for its failure is not known with certainty. It may be that the theoretical basis advanced for it is false, but the unmistakable initial improvement in both cases seems against this conclusion. Perhaps it is more likely that the auricular appendage has failed to become fixed in the new position because endocardium has not fused with endocardium, and that it has therefore gradually escaped from the sutures and retracted out of the valve orifice. Only postmortem examination will show if this has occurred, and both patients are still alive. It is hoped that despite these failures, this report may yet be a stepping stone on the difficult path to successful surgery for mitral regurgitation.

Vagotomy and Gastroenterostomy in the Treatment of Duodenal Ulcer. K. R. Trueman and D. L. Kippen. Canad. M. A. J. **70:**514-517 (May) 1954 [Montreal, Canada].

From May, 1946, when vagotomy in combination with posterior gastroenterostomy was first used at the Winnipeg Clinic, until December, 1951, 1,603 patients with duodenal ulcer were observed, and 377 (23%) of these submitted to surgery. Partial gastric resection was done in 247, vagotomy plus gastroenterostomy in 79, vagotomy alone in 30, and vagotomy plus gastric resection in 21 cases. The 79 patients on whom vagotomy plus gastroenterostomy was done were regarded as intractable, the majority being referred through the medical department for surgery. The average duration of symptoms exceeded 12 years. Hemorrhage occurred on one or more occasions in 35 patients, 19 of whom described hematemesis in addition to melena. Perforation was reported in 15 cases, of whom eight had also bled. Obstruction, as evidenced radiologically by narrowing of the duodenal channel, with marked gastric retention was present in 25 patients. Fifty-one of the 79 patients were interviewed and examined at regular intervals after operation, but questionnaires were sent to all 79, and 72 of these were answered. Of the remaining seven patients, three are known to have died and only four could not be traced. Good results, which imply absence of ulcer recurrence or gastrointestinal symptoms of any consequence, return to normal weight, and ability to resume the previous occupation were obtained in 56 patients. Fair results, that is, freedom from ulcer distress or recurrence, but the presence of slight or moderate side-effects of the operation such as bloating or diarrhea, were observed in seven patients. The remaining nine patients had poor results, having symptoms indicative of recurrence, such as typical ulcer pain; hemorrhage; signs of a crater in the roentgenogram; severe side-effects, such as frequent vomiting or diarrhea; marked restriction of diet or regular use of antacids; and loss of weight and strength or inability to resume previous occupation. Since those classed as fair were quite satisfactory from the standpoint of ulcer control, the diathesis of the duodenal ulcer appears to have been satisfactorily controlled in about 85% of the patients who had vagotomy.

Mammary Cancer Treated by Bilateral Adrenalectomy. L. N. Pyrah and F. G. Smiddy. Lancet **1:**1041-1047 (May 22) 1954 [London, England].

Twenty-two patients with advanced mammary carcinoma with metastases underwent bilateral adrenalectomy and bilateral gonadectomy. One died soon after operation, and 8 either died from cancer or showed no improvement; 10 improved, and 5 of these had a major remission, with great improvement in the general health and with demonstrable regression of the primary growth (if still present) and its visible metastases, and with well-marked radiographic improvement of osseous and thoracic cancerous deposits. It is not known whether these regressions are permanent. The authors' results confirm the work of Huggins and his co-workers that a favorable response sometimes follows bilateral adrenalectomy and oophorectomy. Tumors having an alveolar or adenomatous structure appear to respond most favorably to this treatment; but it appears to be unwise to rely on a single biopsy before rejecting a case as unsuitable for adrenalectomy.

Fat Embolism: A Clinical and Experimental Study of Mechanisms Involved. R. L. Swank and G. S. Dugger. Surg., Gynec. & Obst. **98:**641-652 (June) 1954 [Chicago].

Swank and Dugger say that they have become increasingly aware in recent years that cerebral fat embolism may occur early after injury and without pulmonary symptoms. These patients may have cerebral concussion, complicating fractures, or large area body mutilations, and may recover consciousness in a relatively short time, only to become stuporous again in a few hours. The entire course of these events may take no longer than 6 to 12 hours. These patients subsequently recover or die in 24 to 48 hours. Gross brain lesions are frequently absent at autopsy, but in some cases fat emboli can be demonstrated particularly in the brain and lungs. The authors cite two patients who had been injured in automobile accidents. The manifesta-

tions of fat embolism were primarily cerebral in both cases. In the first case progression to death was relatively fast, but in the second case a temporary stabilization occurred after early cerebral embolism, and this was later followed by pulmonary distress and manifestations of cerebral involvement and death. These early manifestations of cerebral embolism are frequently overlooked because it is generally held that the clinical manifestations of fat embolism are delayed for 48 to 72 hours and are at first pulmonary. Only after the development of shortness of breath, hemorrhagic sputum, and rales at the bases of the lungs is it thought that signs of cerebral embolism develop, namely excitement, stupor, convulsions, paralyses, and coma. The authors present experiments on rabbits, which were carried out to explain the early onset of cerebral manifestations in some cases of fat embolism in which major fractures and head concussion or anesthesia coexist and to clarify some of the factors involved in the mobilization of the fat forming the emboli. These experiments suggest that an important source of the fat emboli is circulating fat globules, which are held much of the time in the capillaries of the general and pulmonary circulation. The early onset of cerebral symptoms of fat embolism is due probably to either shock and unconsciousness or to anesthesia, all of which presumably relax the vascular bed and allow the circulating fat plus the fat from the fractured bones to be washed through the pulmonary circulation to the brain. It is suggested that dark-field examination of whole blood, to determine the amount and sizes of the circulating fat globules in the blood, would be a useful adjunct to the early diagnosis of fat embolism.

NEUROLOGY & PSYCHIATRY

Relationship of Retrobulbar Neuritis to Multiple Sclerosis. R. G. Taub and C. W. Rucker. Am. J. Ophth. **37**:494-497 (April) 1954 [Chicago].

An episode of acute retrobulbar neuritis is recognized as a possible primary symptom of multiple sclerosis. A follow-up study was made in 1953 of patients with acute retrobulbar neuritis of undetermined origin who had been seen at the Mayo Clinic in the period 1937 to 1942, inclusive. This gave an interval of 10 to 15 years after the episode of acute retrobulbar neuritis. Twenty-five of 118 patients with acute retrobulbar neuritis could not be located, and 6 others had died before 10 years had elapsed after their bout of retrobulbar neuritis, but in none of these 6 had signs of multiple sclerosis been evident before death. The 87 patients available for study were divided into three groups on the basis of age. There were seven patients less than 20 years old, and multiple sclerosis developed in one of these. Fifty-seven patients were between 20 and 44 years old, and in 26, or 45%, multiple sclerosis developed. The time between the initial bout of retrobulbar neuritis and the development of symptoms of multiple sclerosis could be determined in 23 of the 26 cases and ranged between one and 14 years. In more than half of the 23 cases, the onset of multiple sclerosis fell within the first five years. Multiple sclerosis developed in only one of the 23 patients of the third group, who were more than 44 years old. Thus the data indicated that, if a person between 20 and 44 years has an attack of retrobulbar neuritis, there is a 40 to 50% chance that multiple sclerosis will develop within 10 to 15 years. In the cases in which retrobulbar neuritis occurs before the age of 20 years, the chance that multiple sclerosis will appear is less likely, and, when it occurs after the age of 44 years, the chance is remote.

Pain in Paraplegia: Clinical Management and Surgical Treatment. E. H. Botterell, J. C. Callaghan and A. T. Jousse. Proc. Roy. Soc. Med. **47**:281-288 (April) 1954 [London, England].

Spinal cord injury is followed by diminished or absent sensation below the level of the lesion. Nevertheless, pain is frequently referred to the portion of the body rendered anesthetic. Botterell and his associates investigated pain in 125 patients with traumatic paraplegia who were treated in a Veterans Affairs neurosurgical unit in Toronto, Canada. A questionnaire was filled out by each, and 34 were interviewed personally. Patients who suffer most severely describe their pain as "sharp," "a dull ache," or "vise-like." This is commonly superimposed on a background of burning pain. The pain in paraplegia is referred to the dermatome corresponding to the injured segment of cord or the anesthetized area below it and is described by the patient on the basis of past experience of pain. One hundred eighteen of the 125 patients with traumatic paraplegia complained of pain. Twenty-nine of the 38 patients who had pain of troublesome severity were able to leave the hospital, although the severity of their pain interfered at times with sleep and appetite as well as with vocational and diversional activities. Some members of this group may yet require surgical treatment. The incidence of severe pain is low in cervical injuries, higher in thoracic and complete conus or cauda lesions, and is commonest after partial destruction of the cauda equina and conus medullaris. Patients may suffer severe pain at the moment of injury or as soon as consciousness is regained and are never free of pain thereafter. In other cases, the onset of pain may be delayed for years. Therefore, in those patients who are at present pain-free, central pain may develop. Distention of the bladder by excessive residual urine, bladder spasms resulting from infection or calculi, and constipation producing distention of the large intestine were local conditions enhancing pain. Successful treatment requires an understanding of the patient's emotional and psychological, as well as physical, requirements. If these needs are met, the majority of patients learn to live with their pain. Nine patients had to be relieved by surgical means. Bilateral spinothalamic tractotomy at the level of the first or second thoracic vertebra or higher proved most efficacious in relief of pain. The histories of six patients who underwent this treatment and who have been followed for from two to eight years are reported. One patient underwent bilateral frontal lobotomy and has been followed for five years. He has retained the ability for planned thinking, a good sense of humor, and concern over the welfare of his family.

Hypnotic Treatment of Phantom Sensation in 100 Amputees. C. Cedercreutz. Acta chir. scandinav. **107**:158-162 (No. 2-3) 1954 (In English) [Stockholm, Sweden].

Hypnotic sleep was given a therapeutic trial in 92 men and 8 women with phantom limb and accompanying phantom sensations after amputation. Of the 100 patients, hypnosis with posthypnotic anesthesia (complete insensibility of the stump after awakening) was achieved in 19 and hypnosis without posthypnotic anesthesia in 26, while the remaining 55 patients could not be hypnotized. Deep as well as light hypnosis affected the phantom sensation in the desired direction. The number of hypnotic sessions required to eliminate the symptoms varied from one to 23. Freedom from symptoms or considerable improvement was obtained in all patients in whom posthypnotic anesthesia was achieved. The patient's age, sex, the location of the phantom, or the period elapsed between the amputation and hypnotic treatment did not exert any effect on the results, which were favorable in 35 patients. Twenty-two of these obtained complete relief from symptoms with disappearance of pain and phantom; pain subsided but the phantom did not vanish completely in 5; appreciable easing of pain and lasting changes in the shape of the phantom occurred in 8. Positive results thus were obtained in more than one-third of the patients. Several of these patients are still free from symptoms three years after completion of the treatment. Late relapses after hypnotic treatment occurred in 12 patients. If the phantom responded quickly to treatment and subsided after two to three hypnotic sessions, long-lasting effects were mostly achieved. If the phantom underwent regressive changes in the course of the treatment or if more than seven hypnotic sessions were required for its elimination, the incidence of relapse was high. This also applies to those cases in which the phantom could not be eliminated entirely.

Memory Disturbances in Third Ventricle Tumours. M. Williams and J. Pennybacker. J. Neurol., Neurosurg. & Psychiat. **17**:115-123 (May) 1954 [London, England].

One hundred eighty patients with verified intracranial lesions were interviewed by psychologists in a neurosurgical unit in order to ascertain whether there is any area of the brain the

destruction of which is specifically associated with impairment of memory. Four cases in which impairment of memory and site of lesion were clearly defined and well localized are described in detail. The incidence of memory disturbances in tumors of the third ventricle was compared with that in other localized intracerebral tumors. Patients with obvious and clear-cut defects of memory were examined for the commonest site of lesion. All analyses support the conclusion that memory impairment is most common and specific when the area surrounding the floor and walls of the third ventricle is disturbed. Lesions further forward, in the frontal area, often result in the patients not bothering to use their memories, while lesions of a cerebral hemisphere may cause loss of memory for particular skills, but the patients remain capable of remembering events; lesions of the posterior fossa may make the patients retarded and depressed and unable to think of remembering. With lesions in and around the third ventricle, however, the patients frequently retain good personality and intellect but show gross defects of memory. The loss is not for particular events, nor for immediate appreciation of impressions, it is rather for their normal endurance and retention. Apart from the site of the lesion, however, it is possible that other factors may be involved in determining the memory disturbance.

Results of Radioactive Isotope Encephalography in Patients with Verified Intracranial Tumors. L. Davis and T. Craigmile. J. Neurosurg. **11**:262-267 (May) 1954 [Springfield, Ill.].

The results of isotope encephalography, performed after the administration of radioactive diiodofluorescein or radioactive sodium iodide in 200 patients with verified primary or metastatic intracranial tumors, are reviewed. Primary intracranial tumors were found in 174 patients, while metastatic neoplastic lesions were encountered in 26 patients. The original end-window Geiger-Mueller counter was used in the majority of the studies, but recently a more nearly accurate scintillation counter was used. No attempt was made to classify the results on the basis of the counting device used. Analysis of these results reveals a slightly higher incidence of accurate findings after the use of diiodofluorescein. In patients with primary intracranial tumors, the use of this substance resulted in accurate localization of the later verified lesion in 61.6% of the patients examined. The use of radioactive sodium iodide gave a precise indication of the site of the neoplasm in 52.8% of the patients examined by that method. In 21 patients with metastatic intracranial tumors, studies following administration of diiodofluorescein showed evidence of the presence of an organic lesion in about 76%. Precise localization of a solitary metastatic nodule was obtained in 47.6% of the patients examined. The results of the examination following injection of radioactive sodium iodide in five patients with metastatic lesions were far from encouraging. One person with a solitary nodule displayed a generalized increase in radioactivity without evidence of focal concentration. All others similarly examined showed no abnormalities in their tracings.

Nocardiosis of the Central Nervous System. E. G. Krueger, L. Norsa, M. Kenney and P. A. Price. J. Neurosurg. **11**:226-233 (May) 1954 [Springfield, Ill.].

A case of pulmonary nocardiosis with development of a metastatic cerebral abscess is reported. The patient, a 42-year-old Negro man, underwent successful excision of a left frontal abscess and received penicillin and chlortetracycline (Aureomycin) in the postoperative course. Complete remission ensued and has lasted 15 months after surgery and 13 months after discontinuance of antibiotic therapy. Tests run on the Nocardia isolated from the abscess showed the organism to be sensitive to chlortetracycline. This is the 20th case of nocardiosis of the central nervous system to be reported. The patients range in age from 20 to 65 years, and men are affected oftener than women. In general, neurological symptoms begin several months after the onset of extraneural manifestations such as cough or subcutaneous abscesses. Clinically, no distinct entity exists; neurological signs are related to localization within the central nervous system and to increased intracranial pressure. Pathologically, focal abscesses are much commoner than primary meningitis. In almost all the autopsies there were additional visceral lesions predominantly in the lungs and the subcutaneous tissues. The authors believe that, in a case of nocardiosis with a cerebral abscess, early surgical removal accompanied by intensive administration of sulfonamide drugs and of broad spectrum antibiotics is the treatment of choice.

On the Inheritance of Epilepsy. O. P. Kimball. Wisconsin M. J. **53**:271-276 (May) 1954 [Madison, Wis.].

Kimball made his studies mostly at the White Special School for Epilepsy in Detroit, the first school of its type in the United States, which was established in 1935. He also investigated cases in his private practice. He explains his point of view on the basis of the history of a family, one child of which attended the aforementioned school. It was learned that his mother had seizures during childhood and adolescence; but when she was 17 the seizures stopped. She had eight living children, and only one had seizures. The electroencephalograms of the father and three children were normal. The mother and five children had electroencephalographic records indicative of epilepsy; yet only the one child had seizures. The author applies the term "potential epilepsy" to the other four children who had the "genetic potential" for epilepsy, but not clinical seizures. When environmental changes occur in the person with potential epilepsy clinical seizures may start, and potential epilepsy may become real epilepsy. The author feels that this combination of genetics and environment is responsible for the existing confusion. That epilepsy is hereditary is shown by the high degree of concordance in monozygotic twins and by the fact that the incidence of epilepsy among the relatives of epileptics decreases as the relationship becomes more distant. The analysis of 520 sibships in which at least one sib was epileptic is the main purpose of this report. Every history was taken, and the follow-up notes were made by the author through a continuous study for over 20 years. Before the special school in Detroit opened, the author found what every student of epilepsy has found in attempting to obtain family histories, either the family did not know, or was unwilling to admit an inherited tendency to epilepsy. Repeated attempts over a period of years frequently produced an accurate history. The attitude of the parents changed when they learned that a special school was ready to accept their child, transport him to and from school, care for him, provide adequate educational facilities, and give medical treatment. In a table that lists the results of investigations on 294 families in which one parent was epileptic, and in which there was a total of 1,070 children, there were 399 epileptic children (37.3%). In 222 families, in which neither parent was epileptic, but in which epilepsy was in the families, there were 772 children, of whom 274 had epilepsy (35.5%). In four families with 25 children, in which both parents were epileptic, 20 or 80% of the children were epileptic. The genealogy of several families with epilepsy is discussed; one, going back to 1630, showed the occurrence of epilepsy in almost every generation, but there had never been a case of epilepsy in childhood, convulsions always started at middle life, usually in the early 40's. The women thought that their epilepsy was due to menopausal change. The author's patient with this type of epilepsy was 42 years old when nocturnal convulsions started. What is to be the answer when a person with epilepsy seeks advice about marriage and having children? If the diagnosis is brain injury with convulsions, then there is no reason for worry over heredity. However, if the diagnosis is genetic epilepsy, he should be told that his disease could be, and probably would be, passed on to his children. In fact, more than one-third of children of epilepsy patients are themselves affected. Even if the children are not so afflicted, the grandchildren might be.

Serology of Multiple Sclerosis. E. Frick. Klin. Wchnschr. **32**:450-452 (May) 1954 (In German) [Berlin, Germany].

Frick points out that Sachs and Steiner reported on serologic studies in multiple sclerosis in 1934, using as antigen an alcoholic extract from the brain of patients who had died of multiple sclerosis. They performed complement fixation tests in 289 cases and obtained positive reactions in 41%. They had assumed

that this serologic reaction represented an antibody function against an infectious organism or against a pathological substance in the central nervous system. These investigations were forgotten until Frick resumed the studies in 1951, closely adhering to the method employed by Sachs and Steiner. Roemer, Schrader, and Schild reported two recent studies yielding positive results in about 50% of 60 patients with clinically established multiple sclerosis, particularly in those with acute exacerbations. In order to establish the specificity of the reaction they examined the serums of 159 patients with other neurological disorders as well as of 269 patients with internal diseases. In the patients with neurological diseases 3.1% showed positive reactions and of those with internal disorders nearly 10%, but in the latter the serum lability reaction was also usually positive. Frick recently made the test on 51 patients with multiple sclerosis. The result was positive in 25 of these patients, doubtful in 5, and negative in 21. Positive reactions were more frequent in the acute than in the chronic forms, but positive results were observed also when acute exacerbations appeared in chronic cases. The author used as antigens formaldehyde-fixed cerebral and spinal tissues of patients with multiple sclerosis, in whom the diagnosis had been established by histological examination. The technique employed was that of the complement-fixation method. Six antigenic extracts yielded largely identical results. Frick found that antigen prepared from normal brain tissue produced equally good results. He feels that the final evaluation of the serologic findings is impossible as long as the antigen has not been chemically analyzed. The suggestion of Roemer, Schrader, and Schild that the antigen is a lipoid or a lipoprotein is shared by Frick. He doubts, however, that it is a substance that occurs exclusively in the brain of patients with polysclerosis, and feels that as yet the serologic reaction cannot be employed to solve etiological or pathogenetic problems of multiple sclerosis. His preliminary conclusion is that the described method permits the demonstration of antibodies in patients with multiple sclerosis and that the presence of these antibodies depends on the state of activity of the disease process. By inactivating the serums at 60 C (140 F) the specificity of the results could be greatly improved and lability reactions simulating positive results could be excluded.

GYNECOLOGY & OBSTETRICS

Morbidity in Vaginal Hysterectomy. J. H. Pratt and Q. Scherman. Am. J. Obst. & Gynec. **67**:1323-1337 (June) 1954 [St. Louis].

A four year study was made of 514 patients undergoing vaginal hysterectomies with or without vaginal repair. The distribution of patients was comparable in the four years with regard to number, age, and association of repair, but the preoperative preparatory routine was changed each year and the various methods compared in relation to postoperative morbidity. The criterion for morbidity was temperature elevation above 100.4 F on any two days following the day of operation; the authors realize this is far from an ideal standard, but it is objective and has been used in other similar studies. The intravaginal use of a suppository containing 100,000 units of penicillin the night before operation was the most effective means of reducing morbidity of any of the methods tried. The introduction of 5 gm. of sulfathiazole into the peritoneal cavity was helpful but the increase in morbidity after the elimination of this procedure from the routine was not marked. In patients on whom hysterectomy and vaginal repair were done, the combined use of penicillin and sulfathiazole reduced the morbidity the most; this effect was primarily on patients 50 years old or less. pHisoderm, a detergent cream composed of sulfonated ether, petroleum, lactic acid, and wool fat cholesterols, was helpful in reducing morbidity but not so effective as the vaginal suppositories of penicillin. The number of serious complications in this series was encouragingly low; however, five patients had to be operated on again within a few hours because of retroperitoneal hemorrhage. There were also 10 patients with vaginal bleeding of mild to moderate degree and 11 others with hematomas of the vaginal vault or the deep pelvic spaces. There were two cases of thrombophlebitis of the deep veins and three of the superficial veins and one of questionable thrombophlebitis. No cases of allergy were encountered in the year in which the routine use of sulfathiazole was discontinued. Urinary retention was numerically the biggest single problem encountered in this series of patients, but, although it caused additional days in the hospital, few elevations in temperature could be blamed on it alone. The variation in vaginal preparation had no effect whatever on the stay in the hospital. Patients with vaginal repairs averaged three days longer in the hospital than those without. Patients who had morbidity were one day longer in the hospital than patients without. Hospital stay averaged one or two days longer for older patients than for younger. However, morbidity was less frequent among the women past 60 years of age. These patients usually had vaginal repair but their uteri tended to be small and atrophic. Loss of blood was less in the older women, the operation went faster, and the anesthetics were of shorter duration. All of these things are factors in lowering morbidity.

Removal of 184 Pound Ovarian Tumor, and Observations Regarding Splanchnic Shock. D. H. Eames Jr. Am. J. Obst. & Gynec. **67**:1358-1364 (June) 1954 [St. Louis].

The removal of a 184 lb. (83.5 kg.) serous and pseudomucinous cystadenocarcinoma of the left ovary of a 44-year-old woman, the 13th largest ovarian tumor reported in the literature, gave the author a chance to observe the effect of sudden release of acute intra-abdominal pressure. It was decided to remove the tumor whole, because tapping and draining represents a danger of allowing malignant cells to escape within the peritoneum. The operation lasted three hours. The lowest depression of the patient's blood pressure was to 78/48 mm. Hg and occurred during the time she was being moved from the left lateral to the supine position for closure of the incision. This is not an unusual reaction and was reversed on cessation of manipulation. The author is of the opinion that splanchnic shock caused by removal of large abdominal tumors is by no means constant in its appearance and is rather uncommon. When it does appear, it is usually manifested some hours after the release in pressure, as a rule 4 to 24 hours later, and the use of "slow" decompression is poor prophylaxis. Instead, the most logical course of action seems to be the removal intact of such large tumors in order to face the possibilities of reaction with the patient in optimum physical state. With the use of whole blood and modern drugs this possibility has become secondary to the evils accompanying the drainage of any large ovarian tumor, and size alone should no longer be considered an indication for drainage rather than intact removal.

Neurogenesis in Ovarian Tumor. H. Selinke and E. Pumares Mogimes. An. Fac. méd. Montevideo. **38**:337-342 (Sept.-Oct.) 1953 (In Spanish) [Montevideo, Uruguay].

A woman 32 years old had a simple ovarian teratoma removed. The operation was followed by development of intraperitoneal metastases and cachexia. At the end of six months after the operation the abdomen of the patient was larger than that of a woman at full-term pregnancy. In a second operation multiple metastases were removed from the peritoneal cavity. The removed tumors were encapsulated, pedunculated, and independent from one another. They contained typical teratoma tissue and a great number of large masses of cerebroid tissue, enclosed in a fibrous meningeal-like membrane. Histological preparations of the cerebroid masses consisted of lobules lined with ectodermal epithelium, neuroblasts in different stages of differentiation and adult neurons, either normal or in a process of degeneration. The stage of development of the nervous tissues was clearly identified with the stains of Bielschowsky and of Rio Hortega. The patient died. A necropsy was not permitted. The authors believe that the histogenesis of neural tissue in teratoma depends on the intratumoral presence of archenteron-like structures that function as archenteron functions in normal embryos. The former in teratoma and the latter in normal embryos induce differentiation of tissue with consequent formation and development of nervous tissue. The tubules lined with ectodermal epithelium

observed by the authors in histological preparations of cerebroid tissues appear to have been the archenteron-like structures in the case reported.

PEDIATRICS

Studies of Serum Electrolyte Changes During Exchange Transfusion. G. Miller, A. B. McCoord, H. A. Joos and S. W. Clausen. Pediatrics **13**:412-418 (May) 1954 [Springfield, Ill.].

Eight infants with erythroblastosis fetalis were treated by exchange transfusion within the first 36 hours of life and were studied in relation to serum electrolyte changes. It was established by tests that hyperkalemia was induced in two of these patients and aggravated in one in whom it was already present. There were no clinical signs or symptoms of hyperkalemia. The elevated plasma potassium content of citrated whole blood following prolonged storage may produce hyperkalemia in some infants. It is suggested that hyperkalemia and hypocalcemia may coexist in some of these babies during exchange transfusion. This potential risk can and should be minimized by the use of relatively fresh blood and the judicious administration of calcium during replacement transfusion with citrated whole blood.

Viral Hepatitis in Early Infancy: Report of Three Fatal Cases in Siblings Simulating Biliary Atresia. R. B. Scott, W. Wilkins and A. Kessler. Pediatrics **13**:447-453 (May) 1954 [Springfield, Ill.].

Three fatal cases of hepatitis with jaundice are reported in siblings with onset at ages of four weeks, one week, and four months and death at three months, four months, and nine months, respectively. In these cases, the disease simulated the clinical picture of biliary atresia. Exploratory laparotomy, performed in all three, revealed biliary atresia in only one; cholecystoduodenostomy was performed and failed to ameliorate the condition. Microscopic findings in postmortem sections of the liver were consistent with a diagnosis of viral hepatitis. From the data available, it appeared to be of the homologous serum type. The mother and two living siblings exhibited laboratory evidence of liver damage. Failure to include a consideration of congenital viral hepatitis in the differential diagnosis of icterus in the very young infant may have serious consequences, since these patients do not withstand surgical procedures very well. The authors feel that the deaths of their three patients, although unavoidable, were perhaps hastened by surgery.

Evaluation of Large Doses of Cortisone in First Attacks of Rheumatic Carditis. E. T. Heffer, R. D. Turin, S. R. Slater, and I. G. Kroop. J. Pediat. **44**:630-639 (June) 1954 [St. Louis].

Nineteen children in their first attacks of rheumatic fever with carditis were treated with cortisone in larger doses than those used by the International Cooperative Study. The initial total daily dose of cortisone was between 200 and 300 mg. given in four divided oral doses. This dose was usually continued for at least two weeks or even longer, until the clinical and laboratory findings indicated maximal suppression of the disease. It was only then that the dose was reduced gradually. On the average 200 to 300 mg. of cortisone was given for two weeks, 150 mg. for one to two weeks, 100 mg. for one week, and 50 mg. for one week. Dosage was individualized according to the severity of the disease and its course. Before treatment was started, all 19 patients had significant organic murmurs; 11 had cardiac enlargement as demonstrated by fluoroscopy and roentgenogram. Fifteen patients had abnormal electrocardiographic findings, and three had heart failure. Of the 19 patients, 13 with monocyclic attacks of rheumatic fever had no residual cardiac involvement after treatment; 10 of these were treated within the first two weeks of their illness. Six patients had residual systolic murmurs after cortisone therapy. These patients were treated late in the disease and either had a long, protracted low-grade activity or a polycyclic course. The immediate suppressive effect of cortisone on symptoms of carditis was remarkable. In three severely ill children, cortisone therapy was considered lifesaving. In one of these patients salicylates had been ineffective. Gallop rhythm usually disappeared within four days, and tachycardia responded to treatment within a week, except in those patients with carditis of several weeks duration before therapy was begun. In several patients, cortisone produced sinus bradycardia and arrhythmia with nodal escape. The prolonged P-R interval usually returned to normal within two weeks. The friction rub that was present in two patients before the treatment disappeared within four days. No new murmurs developed in the 19 patients. There were no complications despite the high total dosage of cortisone employed. The Cushing habitus with moonface, obesity, and striae developed in all patients. Mild facial hirsutism and acne never necessitated cessation of treatment. Elevation of the blood sugar and glycosuria were not observed. Hepatomegaly observed in the first week or two probably represented congestive failure induced by an endogenous water shift secondary to cortisone therapy. Hepatomegaly, which was observed later, probably represented fatty infiltration of the liver. In both cases, there was no contraindication to continued cortisone therapy. The patient with a monocyclic attack of rheumatic fever has a better chance of escaping cardiac damage if he is adequately treated early in the disease. The number of cases reported on is too small for drawing definite conclusions, but the trend shown warrants the use of larger doses of cortisone given early in the disease and continued for a long enough period. In this way it may be possible to prevent residual valvular damage in a monocyclic attack. The authors' experience also indicates that cortisone in adequate dosage may be more effective than salicylates in controlling carditis.

Comparative Study of Children with Bone and Joint Tuberculosis Treated With and Without Tuberculostatic Drugs. M. Modena. Wien. med. Wchnschr. **104**:359-360 (May 1) 1954 (In German) [Vienna, Austria].

The course of bone and joint tuberculosis in 363 children treated by the old methods of sun irradiation and general measures was compared with that in 115 children between the ages of 1 and 12 years treated with streptomycin and *p*-aminosalicylic acid. Dihydrostreptomycin was preferred because of its lesser toxicity. It was given in doses of 0.33 gm. daily to children between the ages of 1 and 6 to 7 years, and in doses of 0.5 gm. daily to children between the ages of 7 and 12 years. The total maximum dose was 80 gm.; the average total dose varied from 30 to 40 gm. The treatment scheme varied according to the location and severity of the lesion. There were only three patients who were resistant to streptomycin. *p*-Aminosalicylic acid was given in doses of 0.2 gm. per kilogram of body weight, and the average total dose was 300 gm. The average duration of the disease was shortened by the treatment with these drugs. Attempts were made at reducing the application of plaster of Paris casts to a minimum. The patients were confined to bed for 6 to 18 months on the average and then were allowed to get up with a supporting apparatus of the type of Thomas' splint. Modena considers it more important to relieve the lower extremities of the body weight, than to immobilize them with plaster casts. Joint function with resulting active mobility, varying from one-third of normal to completely normal, was obtained in 88.4% of the patients with tuberculosis of the hip joint and of the knee joint. The response of the tuberculous process of the bone to the antibiotic treatment was slowest in patients with spondylitis. In the most favorable cases, arrest of the disease occurred about the sixth month, and the first signs of repair were observed between the 10th and 12th months. Plaster corsets should be used less frequently, since the most satisfactory results were obtained exclusively with rest cures with Bradford frames. Of six patients with tuberculosis of the foot treated wtih antibiotics, five obtained two-thirds of their normal mobility and ankylosis occurred in one. Satisfactory healing occurred within 12 months in two patients with tuberculous sacrocoxitis. The earlier the diagnosis, the better were the results. Although tubercle bacilli may not be demonstrated in the early stage of the disease, a therapeutic trial with the tuberculostatic drugs should not be delayed in the presence of a relationship between history and clinical findings.

DERMATOLOGY

Tinea Capitis Caused by Microsporum Canis: Report of an Outbreak in Dunedin Institution. M. J. Marples and E. N. Chapman. New Zealand M. J. **53**:158-162 (April) 1954 [Wellington, New Zealand].

Marples and Chapman describe an outbreak of ringworm of the scalp due to Microsporum canis in the Dunedin Receiving Home of wards of the state. Eight of the 10 resident children were found to be infected. Examination of 92 school contacts failed to demonstrate any transmission of infection. The origin of the outbreak could not be traced. The institution did not own a cat, and an elderly cat belonging to a neighbor showed no evidence of infection. There were a large number of cats, including kittens, living on the grounds of an adjacent empty house. These cats had become very wild and could not be collected for examination. They had the habit of sleeping and sunning themselves in the uncovered sandpit belonging to the institution. M. canis characteristically causes small family outbreaks but not widespread epidemics of ringworm. The transfer appears to be domiciliary and requires close contact. When outbreaks occur in families whose cat or dog can be shown to be infected, it is not possible to discover which of the individual infections represent animal to child or child to child transmissions. In the outbreak described here there was considerable evidence that some child to child transmissions of infection occurred within the institution. It is difficult to believe that all the children concerned were directly infected from cat hairs, since they did not play with the semiwild cats and had not actually handled any cat that could be demonstrated as infected. Furthermore, although infections in all but two of the children were observed within a few days of one another, there were indications at the initial examination with Wood's light that the lesions were not all at the same stage of evolution. The majority of scalp ringworms in New Zealand are caused by the dermatophyte M. canis. This fungus is primarily adapted to parasitic life on domestic pets. Although it can and does infect humans, its virulence is decreased in man. Lesions of the scalp respond to treatment with topical applications and may clear up spontaneously even before puberty. Further, child to child transmissions are not very common, the organism losing its invasiveness after a few human passages. The authors feel that the regulations restricting the school attendance of children with scalp ringworm should be reconsidered, since the close contact necessary for human transmission of infection need not occur in school. The described outbreak shows that, although within the institution child to child transmissions did occur, no transfer of infection to school contacts could be demonstrated.

Isoniazid in Treatment of Lepromatous Leprosy. I. Sigall M. Arq. mineir. leprol. **13**:187-194 (July) 1953 (In Spanish) [Minas Gerais, Brazil].

This is a preliminary report on the results of isoniazid therapy in 18 patients with leprosy of the lepromatous type. The initial dose of 200 mg. was increased at weekly intervals up to a daily dose of 800 or 900 mg. The total dose varied between 44 and 144 gm. in 4 to 10 months. The general condition and the appetite of the patients showed improvement early in the course of the treatment. The number of bacilli in the skin and in the nasal mucosa diminished or disappeared. Marked clinical improvement was observed. Infiltration was controlled, maculas disappeared, the volume of the nodules diminished, erythema subsided, and acute extensive recurrences did not reappear. Satisfactory results from isoniazid alone were obtained in two patients who received the drug for five and eight and one-half months, respectively. In four patients the bacilli became resistant within six or eight months in the course of administration of isoniazid alone. Isoniazid combined with sulfonamides for an additional period of three to five and one-half months gave satisfactory results. The best results of isoniazid treatment were observed in nine patients who received the drug in combination with sulfonamides from the beginning to the end of the treatment, between eight and nine and one-half months. Isoniazid combined with streptomycin was given to three patients for three months. It failed. The combined treatment with isoniazid and sulfonamides for an additional period of seven months gave satisfactory results. The author concludes that isoniazid is of great therapeutic value in lepromatous leprosy. No other drug causes the bacilli in the nasal mucosa and in the skin to disappear so quickly or to diminish so rapidly. No other drug produces a clinical improvement in so short a period of time as isoniazid, alone or combined with sulfonamides.

A Study of Tinea Capitis. W. N. C. Fraser and A. H. Hackett. New Zealand M. J. **53**:162-165 (April) 1954 [Wellington, New Zealand].

A survey of school children in the Auckland metropolitan area revealed that the incidence of tinea capitis is uncommon in the summer months, there being only one case found by Wood's filtered light in a sample of 500 children between the ages of 2 and 12 years. The virulence of ringworm appears to vary somewhat from person to person even though infected from the same animal, and such lesions under the same treatment take varying times to clear up. It appears that most infections are transmitted from animal to human beings, but only rarely and less often if precautions are taken has it been proved to be transmitted from person to person. Living conditions appear to play little part in the transmission of infection, unless animals that may become infected and transmit infection to their human guardians are kept in the family. It appears that both good and poor homes are infected. In the opinion of these authors it is not necessary to keep the child from school; it appears that little or no infection is propagated among school children. Teachers and parents should be made aware of this fact, as valuable schooling is often lost when the child can least afford it.

Progressive Scleroderma and Its Roentgenologic Aspects. E. Nägele and R. Pfister. Klin. Wchnschr. **32**:452-455 (May) 1954 (In German) [Berlin, Germany].

Scleroderma appears in a diffuse or progressive form, a localized or circumscribed form, and as sclerema neonatorum. Nägele and Pfister observed eight cases of the diffuse progressive form. The skin changes begin with a doughy edematous swelling, which becomes transformed into an indurative and then into a sclerotic atrophic stage. The skin becomes tensely drawn, the face mask-like, and fingers and toes stiff and claw-like. The sclerotic changes involve not only the skin but various organs as well; diffuse progressive scleroderma is a systemic disease. The cause has not been fully explained, but it is assumed that a central disturbance of the sympathetic regulation is the basic factor, which, by way of defective nervous, increatory, and circulatory functions, leads to metabolic changes in the connective tissue. Degeneration and dissolution of the elastic fibers is followed by their replacement with collagenous connective tissue. The increase in the thickness of the collagen fibers produces the peculiar hardening of the tissue. The intimal tissue of the small arteries and arterioles becomes saturated with coagulated protein and proliferate. Symptoms suggesting a relation to Raynaud's disease are frequent. Roentgenoscopy may reveal reticulation of both lungs, which is a fibrosis produced by an increase in connective tissue. The resulting dyspnea was formerly ascribed to induration of the skin and musculature of the thorax. The authors cite the case of a woman with severe dyspnea and moderate impairment of swallowing. The differential diagnosis proved difficult. Repeated searches for tubercle bacilli gave negative results. Boeck's sarcoid had been thought of on account of wing-like shadows in the lungs, but sclerodermal changes in the hand skeleton and particularly in the skin and the location of the pulmonary shadows in the lower lobes identified the disorder as scleroderma. In the heart, muscular tissue is replaced by fibrous scar tissue. Roentgenoscopy may reveal a normal heart, but frequently there is hypertrophy chiefly of the right side, most likely the result of pulmonary changes. The digestive tract is frequently involved, particularly the esophagus, in which muscle becomes replaced by connective tissue, and the mucous membrane may become involved secondarily. Difficulty in swallowing is observed in about 50% of the cases, and roentgenoscopy discloses retarded passage through the esophagus with atony and reduced peristalsis. Roentgenologic study of

stomach and intestine also discloses atony and reduced peristalsis. The most important roentgenologic changes of scleroderma are those in the bones. The patients with symptoms resembling Raynaud's disease often show osteolytic changes in the terminal phalanges of the fingers, as well as osteoporosis. Finally, there may appear the characteristic triad: volar flexion of fingers, progressive atrophy of the terminal phalanges, and calcium deposits in the finger tips. Calcium deposits are found also in other parts of the body.

UROLOGY

Peyronie's Disease: A New Approach. G. H. Teasley. J. Urol. **71**:611-614 (May) 1954 [Baltimore].

Teasley discusses a new method of treating for Peyronie's disease (induration of corpora cavernosa), which he first reported in 1952 and which now has been tried in 24 cases. The skin is cleansed with soap and water and a disinfectant. The skin and subcutaneous tissue, down to and around the plaque, are infiltrated with 0.5% procaine solution. A solution of 25 mg. of cortisone, dissolved in isotonic sodium chloride solution containing 0.25% of procaine crystals, is prepared. This will make approximately 1.25 to 1.5 cc. The solution is injected directly into the plaque. It is quite difficult to inject, and unless a Luer-Lok syringe is used the needle will be blown off the syringe. Injections are repeated at intervals of one week. The author used six injections before disappearance of the plaque. When the needle can be felt to scrape against the hard plaque and definitely enter its substance so that injection is particularly difficult, the results are best. Injection about the plaque will produce no complete resolution of the fibrous process. A moderate soreness between injections has been the only complaint. There is no reason to assume that any harmful systemic effect will be caused by cortisone in such a small dosage. Treatment can be repeated if symptoms return.

Renal Function After Complete Bilateral Ureteral Obstruction Following Colporrhaphy. B. R. Harrow. Am. J. Surg. **87**:842-850 (June) 1954 [New York].

Complete bilateral ureteral obstruction occurred a few months after vaginal hysterectomy and vaginal plastic operation in a 56-year-old woman with a recurrent third degree cystocele with a moderate vaginal collapse. After 12 hours of anuria cystoscopy did not reveal any ureteral outflow, and catheters could be inserted no more than 1 cm. into both ureteral orifices. A 20% solution of methiodal (Skiodan) sodium that was injected through an occlusion catheter outlined sharp kinks of both lower ureters. The patient's only complaint was bilateral lower abdominal pain. After 72 hours without urine reaching the bladder, a right nephrostomy was performed resulting in drainage of 2,000 to 3,000 cc. of urine during each of the next two days. The left ureter remained completely occluded. The patient improved rapidly. An intravenous pyelogram performed after 13 days with the right nephrostomy tube clamped demonstrated function in the completely blocked hydronephrotic left kidney. On the 23rd day the patient began to void large amounts of urine presumably from relief of the left ureteral blockage, since the right nephrostomy catheter continued to drain larger amounts of urine. The patient was discharged 36 days after the original plastic surgery, voiding all the urine with the nephrostomy tube constantly clamped. On the 47th postoperative day she was readmitted with fever, chills, left flank pain, and a nonfunctioning left kidney on intravenous pyelography. A left nephrostomy was performed. The nephrostomy tubes were removed on the 68th day, and the patient was discharged 74 days after the colporrhaphy with both wounds healed. Afterward the patient had no urinary symptoms and remained in good health. This report is on the 19th patient in the English literature with complete ureteral obstruction after gynecologic surgery. The patient also represents the fourth such occurrence after an anterior colporrhaphy. It is the fourth proved case of complete ureteral obstruction due to kinking and is the only reported case in which roentgenograms demonstrated the kinks. The most interesting information obtained was that visualization of iodide occurred in a kidney completely obstructed for 13 days; follow-up urograms after four months revealed a normal pyelogram. From a discussion of the experimental and clinical data of renal function and the low secretory pressure continuing in hydronephrosis during complete obstruction, the conclusion was drawn that the main mechanism is glomerular filtration and pelvic and calyceal urothelium reabsorption. The author's case emphasized that kinking alone can produce a complete obstruction that may last for several weeks and thus requires early surgical intervention to relieve the anuria. The rapidity of the hydronephrosis and hydroureter with complete occlusion, the limited extent of the hydronephrosis, the slow reabsorption from the lower segment of the ureter and the lack of flank pain were all demonstrated in the author's patient. The use of the occlusion catheter is invaluable in making an accurate diagnosis in bilateral ureteral obstruction. Bilateral nephrostomy or intubated ureterostomy rather than deligation is recommended as the treatment of choice even after vaginal surgery. Insertion of ureteral catheters in secondary colporrhaphy or in anticipated difficult abdominal surgery is strongly advised as a preventive measure.

Alkalosis as Cause of Prerenal Azotemia: Review of Literature with Five Case Reports. M. K. Dill and A. B. Richter. J. Indiana M. A. **47**:494-502 (May) 1954 [Indianapolis].

When nitrogen retention occurs in kidney failure due to disease processes elsewhere in the body, it is referred to as prerenal azotemia. This does not mean that the kidneys are entirely normal. Prerenal azotemia indicates that the primary disease is outside the kidneys. This disorder causes functional or even structural changes in the kidneys with renal impairment. Prerenal azotemia is common in prolonged vomiting, as in intestinal obstruction and pernicious vomiting of pregnancy, severe prolonged diarrhea, acute infections, congestive heart failure, diabetic coma, Addisonian crisis, alkalosis, and shock. This paper deals with alkalosis as a cause of prerenal azotemia. Five cases of renal impairment or prerenal azotemia caused by alkalosis are presented. Alkalosis may result from the intensive or injudicious oral use of alkalis, such as sodium bicarbonate and calcium carbonate; from vomiting, as in high intestinal obstruction; and from removal of chloride by continuous gastric aspiration. Impairment of renal function with azotemia, creatinemia, hyponatremia, hypopotassemia, hypochloremia, lowered renal function tests, etc., often occurs. This renal impairment is reversible if the alkalosis is promptly treated with infusions of saline solution. Potassium chloride may be necessary. If it is not corrected in severe cases, death may result. The theories advanced in the literature with regard to the cause of renal impairment are reviewed. The authors stress the need for alertness with regard to this serious condition; they feel that it should be oftener emphasized in clinical medicine.

Cancer of the Prostate. C. E. Burford and E. H. Burford. Missouri Med. **51**:443-445 (June) 1954 [St. Louis].

Four hundred eighty men between the ages of 40 and 96 with carcinoma of the prostate were admitted to the department of urology of the St. Louis University School of Medicine. One hundred fifty-five were treated before 1941, i. e., before the neutralizing effect of estrogen was discovered, and 325 were treated after 1941. The operative mortality for the entire series of 480 patients was 2.5%. Of the 155 patients, 82 were operated on without positive pathological findings on palpation, and 73 with positive findings. Eleven (13%) survived for 5 years, 6 (7%) survived for 10 years, and 4 died within 6 weeks after the operation (an operative mortality rate of 5%). Of the 73, 23 (32%) survived for 5 years, 3 (4%) for 10 years and 3 died within 6 weeks after the operation (an operative mortality rate of 4%). Of the 325 patients in whom surgical procedures were employed combined with the use of estrogen, 175 did not have positive pathological findings on palpation and 150 did, with metastases in 38 (25%). Of the 175, 124 (71%) survived for 5 years, 42 (24%) survived for 10 years, and only 1 died within 6 weeks after the surgical intervention (an operative mortality rate of 1%). Of the 150 patients, 90 (60%) survived for 5 years,

29 (19%) survived for 10 years, and 3 (2%) died within 6 weeks after the surgical intervention (an operative mortality rate of 2%). Endocrine therapy has completely changed the treatment of carcinoma of the prostate. Patients with carcinoma of the prostate, regardless of the delay in diagnosis, can be relieved of symptoms with greater safety. Routine rectal examinations at least annually of all men aged 40 years or older offers the best chance for early diagnosis and prolonged survival. Even in advanced cases, a 60% five year survival rate may be anticipated. Testicular evisceration combined with administration of estrogen is the best method of management of carcinoma of the prostate; it offers a 33% 10 year survival rate.

THERAPEUTICS

Trial Treatment of Hodgkin's Disease with Actinomycin. Bertrand-Fontaine, J. Mallarmé, J. Schneider and J. Debray. Presse méd. **62**:737-738 (May 15) 1954 (In French) [Paris, France].

Results obtained by the use of actinomycin C in 15 patients (8 of whom were women) with histologically proved Hodgkin's disease treated at the Hôpital Beaujon-Clichy in Paris convinced those in charge of the experiments that the action of this preparation is clearly inferior to that of the established methods of treatment. The drug, dissolved in water, was first given intravenously in doses increasing gradually from 50 mcg. to 400 mcg. daily; later, the starting dose consisted of 200 mcg. daily and the maximum dose of 400 mcg. was attained more rapidly. The total dosages given were 12,000 mcg. in three patients, 8,000 to 9,000 mcg. in five patients, 7,000 to 8,000 mcg. in two patients, 5,000 to 6,000 mcg in two patients, 3,500, 2,600, and 1,100 mcg. in one patient each. Treatment of the last three patients could not be continued because of serious complications (not necessarily connected with the medication). No parallelism could be found between the size of the total dose given and the results obtained; improvement, when it occurred, always appeared at the beginning of the treatment (during the first week) and was not increased by its prolongation. Anorexia, nausea, abdominal pains, and involvement of the mucous membrane (e. g., stomatitis and pharyngitis) were among the side-effects produced by the drug, which on the whole was poorly tolerated, although no serious accident seemed to be directly attributable to it. Rapid but temporary regression of superficial lymph node swelling was secured in one patient with recent, previously untreated disease, and two others were slightly improved, but in the rest the treatment was without effect. Progressive deterioration resulted in the death of five patients. Puncture biopsy, performed during and after treatment in four, failed to show that actinomycin had any cytotoxic effect. Other therapeutic measures, such as radiotherapy, nitrogen mustard, and cortisone, whether used before or after actinomycin, produced unquestionably better results, both locally and generally.

Manifestations of Phagocytosis in Cases of Allergy to Drugs. L. Meyler. Nederl. tijdschr. geneesk. **98**:1137-1143 (April 24) 1954 (In Dutch) [Amsterdam, Netherlands].

Meyler comments on the L.E. cell in lupus erythematosus, mentioning the factors that Hargraves had listed as essential for the appearance of the L.E. cell. While Hargraves and various other investigators believed that the L.E. cell occurred only in lupus erythematosus, others believed that this cell was found also in other conditions, such as moniliasis and serum disease. Meyler searched for L.E. cells in patients with drug allergy. Although he found no L.E. cells, he observed a manifestation that somewhat resembles the L.E. cell phenomenon; that is, he observed phagocytizing segment-nuclear leukocytes. He cites histories of nine patients in whom allergy developed in response to various drugs, including sulfonamides, arsphenamine, iodine, methylthiouracil, butylethylmalonyl urea (Soneryl), and Peru balsam. The phagocytes were found in the peripheral blood as well as in the bone marrow. The phagocytic cells were mostly leukocytes with segmented nuclei. The phagocytized material consisted of lymphocytes, rarely of leukocytes, and apparently also of thrombocytes and erythrocytes. In several cases both forms of phagocytosis were found in the same patient.

First Results in 12 Cases of Hodgkin's Disease Treated with Actinomycin C. R. Martin and J.-P. Munier. Presse méd. **62**:741-742 (May 15) 1954 (In French) [Paris, France].

Treatment with actinomycin C in 12 patients with demonstrated Hodgkin's disease, most in an advanced and some in a terminal stage of their illness, proved disappointing; 2 of the patients died and only 4 are still in fairly satisfactory condition. Diminution in the size of the enlarged lymph nodes, seen in a few cases, was temporary in character and was apparently due more to the effect of the drug on the surrounding tissues than to its effect on the lymph nodes themselves. In only one instance was it accompanied by a reduction in fever and improvement in the patient's general condition. The treatment was usually well tolerated in the doses given, which consisted of 100 mcg. daily, increased gradually to a maximum of 250 mcg. daily (total dosage at first, from 5,000 to 6,000 mcg., later, 7,000 mcg. and even in one case 9,300 mcg.). The side-effects were slight, consisting chiefly of dizziness and nausea when the daily dosage was increased from 150 to 200 mcg.; occasionally, the injection of the drug was followed by a feeling of general malaise with weakness in the legs. These symptoms regressed within a few days. Icterus appeared in one patient two weeks after cessation of the treatment. Various other forms of therapy, notably cortisone, nitrogen mustard, and x-rays, were tried in some cases with results that, though also temporary, were generally better than those obtained with actinomycin.

The Present Position of Ultrasonics. A. W. Bauer. Brit. J. Phys. Med. **17**:97-102 (May) 1954 [London, England].

Bauer feels that much of the literature of recent years on ultrasonics is concerned with the needless repetition of experiments on small animals with dosages that nobody would think of employing in the treatment of human beings. Great advances have been made in the construction of apparatus. The use of large dosages (more than 3 watts per square centimeter) has been discontinued. There has also been agreement on the use of a frequency of 1,000 kilocycles. The development of the pulsed ultrasonic application has been responsible for great progress. With this method the ultrasonic energy is not applied continuously but in short-term impulses. If, for example, the duration of the impulses, which are repeated 100 times in each second, is limited to one-tenth, we speak about a contact ratio of 1:10, the ratio being that of the duration of the impulse to that of the interval. The heat generated in the course of continuous sonation is almost entirely eliminated by the use of pulsed sonation. The mechanical component of the ultrasonic waves, however, remains active, and the reaction is as if the tissues had been exposed to an uninterrupted mechanical action. Effects due to heat appear either in a very mild form or not at all. Evaluating different views on the mode of action of ultrasonics Bauer directs attention to the predominant importance of the neural factor. The diseases on which ultrasonics exert a favorable action are chiefly those in which the neural factor is of great importance. The results of ultrasonic therapy are discussed on the basis of a questionnaire inquiry made by Stuhlfauth and Woeber in 1953. This questionnaire provided information on the results obtained in more than 100,000 cases. The table that classifies the therapeutic results in percentage figures of very good, good, no or slight effects, and worsening also shows that 28 different entities were treated. The best results were obtained in lumbago, myalgia, contusions, distortions, inflammations of the skin, sciatica, periarthritis, arthritis, and tendovaginitis. Even among patients with Raynaud's disease, epicondylitis (tennis elbow), Sudeck's dystrophy, and Buerger's disease, the combined percentage of very good and good results was between 60 and 70. The author concludes that when used according to proper indications and suitable technique, ultrasonic therapy is effective and free from danger.

Collagen Disease: Scleroderma and Its Treatment with ACTH and Cortisone. G. Marañon and J. Gimena. Ann. endocrinol. **15**:106-115 (No. 2) 1954 (In French) [Paris, France].

Fibrinoid degeneration of collagen, the protein constituent of the connective tissue, is the primary and basic lesion found in all the so-called collagen diseases. This degeneration takes place chiefly in the median and intimal coats of the precapillary arteriole and is followed by lymphocytic and fibrocytic proliferation. The connective tissue reaction may be so intense as to cause diminution or even occlusion of the vascular lumen, thus producing ischemia and even at times osseous atrophy of the kind often seen in scleroderma with sclerodactylia. The cause of the disturbance is unknown, but allergy and hormonal factors are believed to play an important part in its production, and the possibility that other factors, such as infections and toxins of various kinds, may be partly responsible cannot be overlooked. A clear understanding of the relationship between the collagen diseases and the hypophyseal-adrenal system has not yet been arrived at, but corticotropin (ACTH) and cortisone have proved more or less effective in their treatment, producing clinical remissions and even in some cases modifying the underlying histological manifestations of the condition. These effects, however, are usually temporary and cease when the medication is withdrawn. Administration of minimal doses, first of corticotropin and later of cortisone, to two patients in advanced stages of scleroderma with sclerodactylia resulted in improvement that was still evident two months after the cessation of treatment. The first patient, who had associated Raynaud's disease and osteolysis, began to improve within a few days after treatment was started; her movements became easier and more extensive, the flexibility of her skin increased, and she gained weight. Total dosage in her case consisted of 360 mg. of corticotropin given in one 10 mg. injection daily, followed by 950 mg. of cortisone (25 mg. daily). The second patient, who had respiratory system and esophageal involvement and rheumatoid arthritis, received a total of 80 mg. of corticotropin in daily doses of 10 mg., followed by 925 mg. of cortisone in daily doses of 25 mg. Improvement in her case was less than in the first, undoubtedly because the sclerosis was more advanced and because her hands presented severe sclerotic and arthritic deformity. No signs of hormone intolerance were seen in either patient.

Biological Bases for Interrupted Isoniazid Therapy. M. Reale, M. Garaventa and M. Ghione. Minerva med. **45**:801-805 (March 21) 1954 (In Italian) [Turin, Italy].

The incidence and early emergence of isoniazid-resistant strains of tubercle bacilli may be related to the rhythm of administration of the drug. The authors made in vitro studies to determine the bacteriostatic effect of a temporary contact of the tubercle bacillus with the drug. From 3 to 5 mg. of isoniazid per kilogram of body weight was given to the patient with tuberculosis who had not received antibacterial therapy for two days. One hour later, 10 cc. of blood was withdrawn, and, after a few crystals of sodium citrate were added, it was diluted with an equal amount of distilled water. This solution was then put into four test tubes in which plates with smears of sputum of the same patient prepared the day before were immersed. They were then put in a thermostatically controlled environment at 37 C (98.6 F) for two hours, after which the plates were removed, washed with sterilized isotonic sodium chloride solution, put in test tubes containing Youmans culture and placed in the thermostatically controlled environment at 37 C. Readings were made at intervals of 24, 48, 72, and 96 hours. They revealed a 72 hour inhibition of the development of bacterial strains sensitive to isoniazid. This test was made on 19 tuberculous patients. The findings suggested that interrupted administration of isoniazid every three days may, in addition to producing a therapeutic action, defer or prevent the emergence of resistant strains and bring about a more effective and more lasting tolerance to the drug. The authors suggest the following regimen for the treatment of tuberculous patients: a single dose of isoniazid (3 to 5 mg. per kilogram of body weight) every three days; 12 gm. of *p*-aminosalicylic acid in two or three doses the following day; and injection of 1 gm. of streptomycin in a single dose the third day. With this method the total dose of each drug is reduced to one-third without attenuating the curative effect.

Rheumatic Fever Prophylaxis: Control of Streptococcal Upper Respiratory Infection in Cardiac and Rheumatic Fever Patients and Their Siblings; Preliminary Report. R. A. Tidwell. Northwest Med. **53**:470-476 (May) 1954 [Seattle].

All persons with active streptococcic infections of the upper respiratory tract are possible future victims of rheumatic fever. Others, who are symptom-free but whose throat cultures are either persistently or intermittently positive for streptococci, may, as carriers, endanger the health of those with whom they come in contact, especially in school and family groups. The effectiveness of a comparatively inexpensive, long-term prophylactic program for control of streptococcic infection of the upper respiratory tract in patients with rheumatic or cardiac disease and their family contacts was studied in a group of 59 subjects consisting of 31 grade school children, 26 preschool children (many of whom were siblings of those in school), and 2 adults (mothers of children in the group). The series included 11 families and 29 separate children. One 200,000 unit tablet of benzathine penicillin G (Bicillin), a tasteless, long-acting salt of penicillin, was given to each of 58 patients daily, before, after, or between meals, to be swallowed whole with water, chewed, or crushed and taken with food. Two tablets daily were required to control the infection in the remaining patient. All persons took the medicament willingly, and its effectiveness in eliminating the beta hemolytic streptococcus was apparently not influenced by either the time or the manner of administration. No evidence of sensitivity to penicillin or of drug resistance was seen in any of the patients during the trial period of from 3 to 10 months. Acute or chronic illnesses present in 25 patients at the start of the prophylactic program included congenital heart disease, 8; rheumatic fever, 13; nephritis, 2; asthma, 1; and streptococcic pharyngitis, 1. The increase in general health as reflected in improved school attendance was remarkable, even in the children with cardiac malformations. No early or late suppurative or nonsuppurative sequelae followed any of the various intercurrent infections experienced by the patients and in none of those who were ill at the start of the program was there any evidence of recurrent rheumatic fever or bacterial endocarditis or any aggravation of renal disease or asthma. Throat cultures taken at monthly intervals or oftener during the period of treatment remained consistently negative for beta hemolytic streptococci, except in one case in which the organism reappeared temporarily when medication was interrupted and in another in which control was secured by doubling the dose. Long-term prophylaxis with benzathine penicillin G can, therefore, be regarded as an economical and effective method of controlling streptococcic infection of the upper respiratory tract.

Local Ambulatory Treatment of Chronic Leg Ulcers with Hyaluronidase, Plasminogen, and Antibiotics. I. R. Spier and E. E. Cliffton. Surg., Gynec. & Obst. **98**:667-674 (June) 1954 [Chicago].

Sixteen patients with chronic leg ulcers, who previously had been treated by routine methods for long periods of time without success, were treated by Spier and Cliffton with a combination of enzymes and antibiotics. Many of these patients had received surgical treatment. All had good arterial pulses in their feet. Fifteen were treated entirely as outpatients, and the remaining patient was an inpatient who was being prepared for skin grafting. These lesions were regarded primarily as the result of stasis changes; therefore, surgery and all measures capable of reducing this stasis such as elevation, diet, and elastic bandages were utilized. The general management was not changed. The involved area was first washed with a detergent cream composed of sulfonated ether, petroleum, lactic acid, and wool fat cholesterols with 3% hexachlophene solution added (pHisohex). Then, instead of applying the scarlet red, gentian violet, hydrophilic nitrofurazone (Furacin) or other ointments, plasminogen or hyaluronidase with or without antibiotics was used. Treatment of the wound was completed with a sterile dressing and an Ace bandage. The first patients were treated with liquid preparations in the outpatient department five days a week for an initial period, and thereafter three times a week. The enzyme and antibiotic in solution were placed on the ulcer for 30 minutes. Then a gauze pad saturated with the solution was applied over the ulcer and held in place with a strip of petro-

latum gauze, dry sterile dressings, and Ace bandage. At the outset of this study, alternate cases were treated with plasminogen, plasminogen plus oxytetracycline (Terramycin), hyaluronidase, and hyaluronidase plus oxytetracycline. After the initial period, the routine of dressings was hyaluronidase with or without oxytetracycline, three times a week. The last six patients were given the medicaments in a water soluble base to take home. This ointment was to be kept refrigerated and applied daily at home. These patients were seen weekly. For the first week the ointment they received contained plasminogen, with bacitracin in four instances and with oxytetracycline in two. Subsequently, hyaluronidase with the antibiotic in ointment was used for the next one or more weeks until the cellulitis cleared, and then hyaluronidase in ointment alone was applied. This procedure proved successful in many cases in which the customary methods had failed.

Anaphylaxis Due to Topical Penicillin. E. S. Carter Jr. and C. B. Cope. J. Allergy **25:**270-271 (May) 1954 [St. Louis].

A 44-year-old nurse while receiving a heavy metal treatment for congenital syphilis in 1943, had a generalized pruritic rash that disappeared after cessation of treatment. In July, 1946, because of a persistently positive serologic test for syphilis, she received 4,800,000 units of penicillin over a 15 day period without side-effects. In February, 1953, she was given an injection of penicillin for bronchitis, and she noted tightness in the chest and wheezing. Two days later a rash appeared, and, although a provisional diagnosis of measles was made, no other stigmas of that disease developed. On May 26, 1953, she noted slight burning and redness of the right eye. She applied some penicillin ophthalmic ointment. This was quickly followed by shortness of breath, a flushed appearance, cramping abdominal pain, and the passage of flatus and a large watery stool. Asthmatic wheezes were audible throughout both lung fields. The blood pressure was 70/50 mm. Hg. Within 10 minutes of the onset of this reaction she was given epinephrine, phenobarbital (Luminal), atropine sulfate and tripelennamine (Pyribenzamine) and responded favorably. A patch test with penicillin ophthalmic ointment several months later, produced a pruritic, erythematous wheal 1.5 cm. in diameter with several small pseudopodia. The authors feel that if the skin rash, which developed after the second exposure to parenteral administration of penicillin, had been evaluated in its true light and its importance impressed on the patient, she would have been spared the last severe experience. It has been proposed that a patient having survived an anaphylactic reaction be provided with identification to the effect that penicillin not be administered under any circumstances. The ease with which alarming reactions are precipitated by seemingly insignificant doses of penicillin is most distressing, and it is not impossible that injudicious skin testing itself may provoke a severe or perhaps even fatal reaction.

Local Haemorrhage and Necrosis of the Skin and Underlying Tissues During Anti-Coagulant Therapy with Dicumarol or Dicumacyl. H. Verhagen. Acta med. scandinav. **148:**453-467 (No. 6) 1954 (In English) [Stockholm, Sweden].

Thirteen cases are reported of an unusual complication occurring after the administration of bishydroxycoumarin (Dicumarol) or ethyl biscoumacetate (Dicumacyl); they are believed by the author to be caused exclusively by these drugs. The pattern of occurrence was fairly regular. Thrombosis developed in a patient, usually a woman, often after parturition or surgery. Therapy with one of the two drugs was instituted, and, on the fifth or sixth day of treatment, a large hemorrhage suddenly developed in the skin, with a hemorrhagic infiltration of the skin and subcutaneous tissues that was extremely painful and was surrounded by a hyperemic reactive zone. A few days later the infiltrated tissues became necrotic, with hemorrhagic blisters. During the next weeks these necrotic tissues demarcated, leaving a deep ulceration. Finally this ulcer healed, leaving an extensive scar. The lesion was localized to the buttock in five of the patients, the thigh in four, the lower leg in two, the penis in one, and the mammary gland in one. Once a hemorrhage had appeared, the continued administration of bishydroxycoumarin or ethyl biscoumacetate as anticoagulants had no adverse effect on the lesion. The patients received routine wound treatment, and three had skin grafts.

Eight Fatal Anaphylactic Reactions to Penicillin. A. Rosenthal. New York J. Med. **54:**1485-1487 (May 15) 1954 [New York].

In the course of an investigation of sudden deaths conducted by the Office of the Chief Medical Examiner of the City of New York, eight instances of sudden death following the injection of penicillin were found. At autopsy, which was performed in all but two of the cases, a search was made at the site of injection to determine whether any of the drug had entered the blood stream. In none of the cases was there any penetration of a vessel by the needle used to inject the penicillin. A review of the types of penicillin used in these cases and of other penicillin reactions, not necessarily fatal, led to the conclusion that it was not one type of penicillin, nor even the procaine radical incorporated in the penicillin, but rather the penicillin itself that was the sensitizing agent and thus the responsible agent for subsequent reactions. Reports of fatal and nearly fatal reactions to penicillin are rapidly increasing and demonstrate the ever-mounting importance of the problem of penicillin sensitivity. Discrimination must be exercised in the use of this drug. There is indiscriminate application of all antibiotics, but, since penicillin was available before the others, the opportunity for its more widespread use and consequent production of sensitization was much greater. That reactions to penicillin occur after use by other than the injection route is now an established fact. Penicillin should not be given parenterally without first asking whether the patient had ever experienced an eruption, rash, swollen joints, shortness of breath, or blotching of the skin following the use of penicillin. A positive answer or even a suggestive positive answer to such questioning should contraindicate the use of penicillin in any form. The presence of asthmatic symptoms at a time when the use of penicillin is contemplated should also serve as a contraindication to its use. Inquiry regarding previous reactions and the restriction of the use of penicillin to necessary instances will tend to prevent the tragedies exemplified in the eight reported cases.

Complications of Anticoagulant Therapy: Review of the Literature and Report of Case. R. C. Rainie. New England J. Med. **250:**810-812 (May 13) 1954 [Boston.]

The anticoagulants are recognized as an important addition to the therapeutic armamentarium of thromboembolic disease. Although hemorrhage is the only known complication, there are numerous contraindications: blood dyscrasias manifesting hemorrhagic tendencies; large open surfaces with poorly controlled hemostasis after operation; surgery of the brain and spinal cord; active or imminent gastrointestinal ulceration or bleeding; malignant hypertension, particularly when there has been a previous cerebral hemorrhage; and subacute bacterial endocarditis. The most important contraindication—the lack of adequate laboratory facilities—may have disastrous results. The incompatibility of lumbar sympathetic blocks and other deep needle-puncture procedures and anticoagulant therapy has been stressed in several reports. Hemorrhage into the spinal cord has been observed as a result of a diagnostic lumbar puncture. A patient with a fractured hip was given anticoagulants two weeks after operation, with resultant extensive ecchymosis at the site of the pinning. Questionnaire inquiries revealed many unreported cases of hemorrhagic complications occurring during the use of anticoagulants. In a review of 107 cases of fatal drug poisoning, it was found that bishydroxycoumarin (Dicumarol) was the commonest cause of deaths resulting from drugs given orally. The author presents the history of a patient with thrombophlebitis that demonstrates the potential hazards of the anticoagulant drugs. It is reemphasized that a reliable laboratory and a competent physician are essential for correct use of these drugs. Direct effects of the anticoagulants on the vascular wall have not been determined, and individual idiosyncrasies cannot be predicted.

Potentially Dangerous Group O Blood. J. M. Gardner and G. H. Tovey. Lancet **1:**1001-1004 (May 15) 1954 [London, England].

Patients with blood belonging to groups A, B, or AB must often be given emergency transfusions of group O blood, although, whenever such transfusions are given, there is a possibility that the transfused anti-A or anti-B antibodies from the group O plasma may react with the recipient's A or B red blood cell antigens to cause a hemolytic transfusion reaction. Investigation showed that most of the group O bloods containing a significant concentration of immune anti-A or anti-B antibodies can be detected by a simple screening test for anti-A and anti-B hemolysins. The test, which is an extension of the routine test for anti-A and anti-B saline agglutinins used by all major blood banks, can be performed with very little extra work and without any additional materials. Application of the test to samples of serum from 1,960 group O blood donors selected at random showed that 185 contained strong anti-A or anti-B hemolysins. Strong hemolysins are those that produce from 50% to complete hemolysis. Clinical and experimental investigations have not yet determined whether the dangerous component of group O blood is the hemolysin or the immune agglutinin, but apparently if group O bloods containing immune anti-A agglutinins in a titer greater than 1 in 16 can be eliminated, no untoward reaction to immune agglutinins need be feared. Investigation of this factor showed that while immune anti-A agglutinins in a titer greater than 1 in 16 were present in 10% of the group O serums containing strong anti-A hemolysins, no immune agglutinins in this concentration were found in 772 group O serums from which strong hemolysins were absent. Adoption of this simple test for hemolysins as a routine screening procedure for group O donors may be expected to ensure the transfusion of group O bloods containing hemolysins or immune agglutinins (anti-A or anti-B) in high concentration only to patients known to belong to group O.

Urinary Excretion of Adrenocortical Steroids by Patients Receiving Salicylates. M. J. H. Smith, C. H. Gray and J. B. Lunnon. Lancet **1:**1008-1009 (May 15) 1954 [London, England].

The urinary excretion of adrenocortical steroids in patients receiving salicylates was investigated by a paper chromatographic method that permitted separate estimation of compound E, compound F, and tetrahydrocortisone. Three of the patients had rheumatic fever, and two had rheumatoid arthritis. All were given sodium salicylate at 4 hour intervals (total daily dosage, 150 to 200 grains [9 to 12 gm.]); in addition, the two patients with rheumatoid arthritis subsequently received 40 units of corticotropin (Acthar-gel) on two successive days. The results showed that, when the salicylates were being given, the urinary excretion of adrenocortical steroids did not exceed the normal range and that, when administration of the salicylates was stopped, there was no decrease in the excretion of the steroids. Subsequent administration of corticotropin to the patients with rheumatoid arthritis, however, was followed by a considerable increase. These findings indicate that, while there are certain similarities in the metabolic and clinical effects of salicylates and of corticotropin and cortisone, the hypothesis that the therapeutic activity of salicylates is mediated by the anterior lobe of the pituitary and the adrenal cortex is untenable.

PATHOLOGY

Relationship Between Mediastinal Lipomas and the Thymus. M. Rubin and S. Mishkin. J. Thoracic Surg. **27:**494-502 (May) 1954 [St. Louis].

The 19-year-old woman reported on by Rubin and Mishkin was admitted in labor to Morrisania City Hospital. A chest roentgenogram revealed a large mass in the anterior mediastinum to the right of the heart. The patient was discharged after delivery and advised to return after convalescence. On readmission, two months later, the patient stated that shortness of breath had increased in severity. Except for dulness and diminished breath sounds over the right side of the chest anteriorly, no other physical abnormalities were found. Angiocardiographic studies showed the mass to be unrelated to the heart and great vessels. The radiographic appearance of the tumor suggested a mediastinal lipoma. The tumor was removed and was found to contain thymic and fatty tissue. The possibility suggested itself that mediastinal lipomas may originate in the thymus, as a result of the infiltration of the organ with fat during its involution. A review of the literature of so-called lipothymomas (the tumor encountered in the patient in question was representative of this type) and lipomas of the mediastinum gave support for this hypothesis. It was noted that, with few exceptions, intrathoracic lipomas are located in the anterior mediastinum in front of the heart and great vessels. Many of the large lipomas, exceeding the bounds of the mediastinal compartment, were connected by a pedicle or a projection of tissue with the thymic area. Benign mediastinal tumors consisting of an admixture of thymic and adipose tissue are comparatively rare. They are found in children between the ages of 10 and 15 years. The authors assume that the mass in their patient was present when a so-called enlarged heart was found in the roentgen examination of the chest at the age of 14 years. Such tumors may simulate cardiac enlargement. The authors attempt to reconstruct the possible evolution of lipomas or lipothymomas, pointing out among other factors that, if such a tumor is discovered early in life, both thymus and fatty elements may be found, but that, if involution has taken place, fatty tissue predominates.

Thymic Cysts: Review of the Literature and Report of Two Cases. W. G. Krech, C. F. Storey and W. C. Umiker. J. Thoracic Surg. **27:**477-493 (May) 1954 [St. Louis].

Krech and associates report on two men, aged 21 and 26, respectively, in whom thymic cysts appeared as anterior mediastinal masses in roentgenograms. The growths were recognized as thymic cysts on histological examination after excision at thoracotomy. Eight additional thymic cysts excised through a cervical incision or at thoracotomy, or both, were tabulated from the literature. The authors believe that thymic cysts may be divided into three groups: (1) congenital, (2) inflammatory, and (3) neoplastic. The non-neoplastic, noninflammatory cysts of the thymus observed in the two patients reported on here are probably congenital in origin. A congenital defect may have been present in the form of a patent thymic or thymopharyngeal duct persisting until such time as fluid or hemorrhagic distention occurred. Why this happens is not known. Cysts resulting from infection appear to be due to syphilis. Although rather common in the past, thymic cysts of this kind are now rarely observed. Cysts occurring in thymic neoplasms can probably be attributed to degeneration and necrosis of the tumor. The preoperative diagnosis of a thymic cyst is presumptive. Any cervical or anterior mediastinal mass may be found to be such a lesion. The presence of a mass that could be a cyst of the thymus is an indication for its complete excision. This is usually accomplished either through a cervical or thoracic approach, depending on the location of the mass. At times a combined approach may be required in order to remove a thymic cyst that lies partly in the neck and partly in the mediastinum. Thymic cysts may be unilocular or multilocular. The lining epithelium is characteristically cuboidal or pavement-type epithelium. In one of the two reported cases medullary and cortical thymic tissue elements were immediately adjacent to this epithelial lining or separated from it by collagenous fibrous connective tissue of varying thickness. Hassall's corpuscles were present and most of them were calcified.

Biopsy of Kidney in Prone Position. R. M. Kark and R. C. Muehrcke. Lancet **1:**1047-1049 (May 22) 1954 [London, England].

The authors have improved on Iversen's technique of kidney biopsy by placing the patient in the prone position. This is more comfortable for him, and it results in a greater number of successful biopsies. The authors obtained 48 specimens from their first 50 biopsies with Franklin's modification of the Vim-Silverman needle. The two failures occurred in a grossly hydronephrotic kidney and in a small and very mobile kidney in a thin woman. Kidney biopsy is of great value in the clinical and pathological diagnosis of renal disease, and should be employed in that field, since it is a safe and relatively painless procedure.

RADIOLOGY

The Pulmonary Aspects of Polyarteritis (Periarteritis) Nodosa. H. P. Doub, B. E. Goodrich and J. R. Gish. Am. J. Roentgenol. 71:785-793 (May) 1954 [Springfield, Ill.].

Of 15 male and 4 female patients between the ages of 2½ and 64 years with polyarteritis nodosa, who were studied between 1943 and 1952, 11 died. Postmortem examinations were done in nine. Seven of the eight remaining patients are living and without symptoms. Duration of life since diagnosis was made by biopsy was six years in three patients, four years in one, two years in two, and one is a current case. Pulmonary symptoms and roentgenologic abnormalities in these patients were neither frequent nor helpful in diagnosis. In the nine patients in whom necropsies were done, death occurred within eight months after the onset of symptoms. Eight patients died of progressive disease with no remissions; one was well for three years after diagnosis was made by biopsy and then died of an acute recurrence in eight weeks. There were observed pathological lesions of polyarteritis nodosa in all nine cases in one phase or another in a generalized distribution. Seven showed characteristic lesions in either the bronchial or pulmonary arteries, one in both. In all the fatal cases, the appearance was that of progressive chronic illness, with increasing pallor, weakness, and emaciation. All showed evidence of multiple systemic involvement. Those having extensive roentgenologic changes in the lungs were observed to have minimal and incompatible physical findings. Pulmonary symptoms were frequent and handicapping in the patients who died. Analysis of the roentgen changes in the six patients with evidence of pulmonary involvement disclosed the presence of parenchymatous infiltration in all. In two the familiar bat-wing areas of infiltration were demonstrated. Two showed moderately pronounced nodulation. These findings are not specific but may be suggestive. Major pulmonary symptoms or parenchymal roentgenologic abnormalities indicate a poor prognosis.

Roentgenographic Demonstration of Histologically Identifiable Renal Calcification. J. D. Mortensen, A. H. Baggenstoss, M. H. Power and D. G. Pugh. Radiology 62:703-712 (May) 1954 [Syracuse, N. Y.].

Postmortem roentgenographic examinations were made of kidneys from 210 patients in whom renal parenchymal calcification had been recognized histologically at the department of pathologic anatomy of the Mayo Clinic. Of the 210 kidneys that were found to contain microscopic evidence of calcification, 174 (82.8%) did not show radiopaque shadows of any type; in 30 (14.3%), radiopaque shadows were observed in isolated portions of the kidney or in localized pathological lesions involving the kidney. In only 6 (2.9%) were multiple diffuse parenchymal calcifications (nephrocalcinosis) demonstrated roentgenographically. An attempt was made to correlate the roentgenographic demonstrability of deposits of calcium with their histopathological features, chemically determined quantity, and associated causative disorders. The following conclusions appeared to be justified. Parenchymal calcification commonly exists in kidneys to a degree, extent, or type that is not discernible roentgenographically. In certain instances renal calcification that histologically is not readily distinguishable from that which cannot be demonstrated roentgenographically does cast characteristic and identifiable shadows on the roentgenogram. The cases in which the authors were able to demonstrate the presence of calcium deposits roentgenologically were associated with primary hyperparathyroidism, chronic pyelonephritis, and chronic glomerulonephritis as causative disorders. This group of cases did not include specimens of nephrocalcinosis caused by hyperchloremic acidosis, recurrent renal lithiasis, or hypercalciuria of undetermined cause, which comprise the only other causative disorders known to be responsible for producing roentgenologically demonstrable nephrocalcinosis. The reasons why some renal parenchymal calcification is and some is not apparent roentgenographically are not clear and need further study. The quantitative chemical determinations in general indicate a higher content of calcium in those kidneys with roentgenologically demonstrable deposits. On the other hand, neither gross nor microscopic estimation of degree of renal calcification is indicative of its roentgen demonstrability. Roentgenography appears to be a practical method for separating a small group with a certain degree or type of calcification from the large group with microscopically demonstrable renal calcification. This small group has certain clinical implications that do not apply to other patients having renal calcification. The term "nephrocalcinosis" should be reserved for this type or degree of renal calcification that can be shown roentgenographically.

Irradiation Therapy in Hodgkin's Disease. C. M. Nice and K. W. Stenstrom. Radiology 62:641-653 (May) 1954 [Syracuse, N. Y.].

In irradiating patients with Hodgkin's disease, as well as with other malignant conditions, it is essential to attempt to utilize an optimum dosage within certain time limits. The treatment should be individualized. At the department of radiology, University of Minnesota Medical School, in general, when the disease was localized to one or a few regions, an attempt was made by the authors to deliver a minimum of 2,000 tissue roentgens to the tumor in 14 days. In the cervical region, this was accomplished by giving 900 r in air to each of three fields. The technical factors were tension of 220 kv., 0.55 mm. Cu and 1 mm. Al filtration, a half-value layer of 1.35 mm. Cu, and 60 cm. distance. Added filtration and increased distance to achieve better depth dose distribution were used for more deep-seated lesions. Large masses of long standing may require heavier dosage, but, even in the smaller masses, a minimum dose of 2,000 tissue roentgens is desirable. When there was massive involvement of the mediastinal nodes, small doses of 50 to 75 r in air were used initially to obviate possible edematous compression of the tracheobronchial tree. A total tumor dose of 2,000 tissue roentgens was still given within a period of three weeks. Total body or spray irradiation was occasionally given in very small doses to patients with widespread involvement. Of 208 patients with proved Hodgkin's disease followed up for five years or longer, 52 (25%) survived for five years. Of 173 patients followed up for 10 years or longer, 19 (11%) survived for 10 years. Clinical staging is considered as the most accurate aid in prognosis and is necessary in comparing series from various medical centers. Of the 208 patients, 20 were in stage 1 (involvement of only one lymph node region or a single lesion elsewhere, with no constitutional symptoms); 17 (85%) of the 20 patients survived for five years; 13 of these were followed for 10 years or longer; and 10 (77%) survived for 10 years. In stage 2 (involvement of two or more proximal lymph node regions confined to either upper or lower trunk, with or without constitutional symptoms), there was a 90% survival, i. e., 18 of 20 patients followed five years, and a 35% survival, i. e., 6 of 17 patients followed 10 years. Thus the difference between stages 1 and 2 was shown in the 10 year period. In the much larger group, stage 3, representing those patients with disseminated disease, the 5 and 10 year survival rates were 10% (17 of 168 patients) and 2% (3 of 143 patients) respectively. In stage 1, the treatment of choice is either intensive irradiation or, possibly, surgical excision followed immediately by intensive irradiation. In stage 2, intensive irradiation is the procedure of choice. In stage 3, less intense or palliative irradiation to reduce tumor masses or to relieve symptoms is indicated. It is in the latter group that the nitrogen mustards and triethylene melamine may serve as useful adjuncts.

BOOK REVIEWS

Improvement of Patient Care: A Study at Harper Hospital. By Marion J. Wright, R.N., M.S., Associate Director, Harper Hospital, Detroit. This book is published in cooperation with and under sponsorship of American Hospital Association. Cloth. $5.50. Pp. 236, with 4 illustrations. G. P. Putnam's Sons, 210 Madison Ave., New York 16, 1954.

This volume presents a comprehensive study of patient care based on the view that each member of the hospital staff plays an important part in the hospitalization program. Careful studies were thus made in selected areas to determine the extent of hospital needs and the methods that might best be employed to increase the efficiency of hospital operation. Patients were interviewed, as well as members of the medical staff, nurses, and other professional and nonprofessional personnel. Data were likewise obtained with reference to types of patients admitted, degree of illness, average length of stay, occupancy rates, admission and discharge procedures, physicians' orders, number of treatments and medications, and other items. After basic data had been collected on the amount and kind of care required, the principles of industrial engineering were applied with work samplings in many areas to establish time schedules, new patterns of staffing, and more efficient procedures wherever indicated. While this study was related mainly to a selected hospital group, the information gathered and the results obtained should be of great value to any hospital. It is fortunate, therefore, that the author's report has been made available in book form.

Guide to Standards for Resident Camps for Crippled Children. Easter Seal publication no. E-11. Paper. $1.50. Pp. 29, with illustrations. National Society for Crippled Children and Adults, Inc., 11 S. La Salle St., Chicago 3, 1954.

This booklet, compiled for the use of those concerned with orthopedically handicapped children, sets forth standards in relation to (1) physical plant and equipment, (2) admission policies and procedures, (3) health and sanitation, (4) safety, (5) administration, (6) program, and (7) personnel of resident camps. In the appendix suggestions are given concerning the groups and individuals who may cooperate in a camping program for orthopedically handicapped children. The appendix also deals with the manner in which national, state, and local resources may be used in establishing and operating such a camp and with some of the legal and insurance problems in camps for crippled children. The detailed recommendations given for each of the categories mentioned are logically grouped under the various aspects of the camping situation within that division. For example, under health and sanitation, standards are suggested for medical examination of campers and staff, health precautions, food, and sanitation. This plan makes quick reference to a particular phase of camping possible and at the same time draws attention to the need for control of all the factors involved in the camping program.

The recommendations made go beyond what might be termed minimum standards; they include some that should be met immediately without qualification and others that are desirable goals but not essential to health and safety. No apparent differentiation is made between these two types of standards. While strong arguments can be offered against the presentation of minimum standards alone, it would seem that, since both types are included, some distinction would be helpful to those using the book. In certain instances the standards are less specific than some might wish to have them; however, reasonable flexibility to permit local adaptation within such standards is desirable. Guidance on specific provisions is available for the asking in most instances, from appropriate national, state, and local agencies. This guide should be extremely helpful to those concerned with the operation, direction, and design of camps for handicapped children.

These book reviews have been prepared by competent authorities but do not represent the opinions of any official bodies unless specifically so stated.

French's Index of Differential Diagnosis. Edited by Arthur H. Douthwaite, M.D., F.R.C.P., Senior Physician, Guy's Hospital, London. Seventh edition. Cloth. $20; 105s. Pp. 1046, with 731 illustrations. Williams & Wilkins Company, Mt. Royal and Guilford Aves., Baltimore 2; (John Wright & Sons, Ltd., 42-44, Triangle West, Bristol 8, England), 1954.

It has been nine years since the last edition of this valuable textbook and five years since the death of French, who before his death had already turned the editorship of his book over to his associate. Over half of the first edition published in 1912 was written by French himself; although none of the sections of the new edition bear his signature, the general plan of the book is unchanged. The book has been thoroughly revised, and only 5 of the 18 contributors also contributed to the sixth edition. Unlike most revisions, this one is slightly smaller than the last. Condensation has been accomplished, despite the addition of much new material, by deleting that which has become obsolete, by shortening some of the longer discussions, but chiefly by painstakingly eliminating overlapping in various sections. Even the index in the back has been shortened and simplified. The book is still profusely illustrated with black and white and colored plates; it remains what it has been for years, a most useful reference book for student and practitioner.

Methods of Biochemical Analysis. Volume I. Edited by David Glick, Professor of Physiological Chemistry, University of Minnesota, Minneapolis. Cloth. $9.50. Pp. 521, with illustrations. Interscience Publishers, Inc., 250 Fifth Ave., New York 1; Interscience Publishers, Ltd., 88-90 Chancery Lane, London, W.C.2, 1954.

Because annual reviews are so useful in the fields in which they are available, with this volume Dr. Glick has started an annual review of methods of biochemical analysis. Comparatively few subjects are to be included in each volume, but each will be covered thoroughly by experts selected because they either "originated the methods . . . or have had intimate personal experience with them." The topics covered in the first volume are zone electrophoresis (Kunkel) and determination of sulfhydryl groups (Chinard and Hellerman); phenolic compounds (Bray and Thorpe); antibiotics (Kersey and Fink) and vitamin B_{12} (Hoff-Jørgensen) by microbiological methods; ascorbic, dehydroascorbic, and diketogulonic acids (Roe); adrenal steroids (Haines and Karnemaat) and radioactive iodine compounds from the thyroid gland and body fluids (Roche, Lissitzky, and Michel) by chromatography; choline (Engel, Salmon, and Ackerman); nucleic acids (Volkin and Cohn); raffinoise and ketose in plants (Whalley and Gross); adenosine triphosphate and related products (Strehler and Totter); catalyses and peroxidases (Maehly and Chance); hyaluronidase in vitro (Tolksdorf); serum lipoproteins by ultracentrifugal analysis (de Lalla and Gofman); and urinary neutral 17-ketosteroids (Engel). The subject index seems adequate, but there is no author index, probably because the references in the bibliographies at the end of each chapter are arranged alphabetically by author. The printing and binding are good. The series ought to be extremely useful.

Wine as Food and Medicine. By Salvatore P. Lucia, A.B., M.D., Sc.D., Professor of Medicine, University of California School of Medicine, San Francisco. Cloth. $3. Pp. 149. Blakiston Company (division of Doubleday & Company, Inc.), 575 Madison Ave., New York 22, 1954.

This book deals with wine as a food and medicine, covering such aspects as the chemistry of wine; its action on the gastrointestinal, respiratory, cardiovascular, and neuromuscular systems; its use in acute infectious diseases, in diabetes mellitus, and in the treatment of the aged and the convalescent; and its employment as a vehicle for medication. The interested reader will find numerous useful references.

QUERIES AND MINOR NOTES

ROUTINE USE OF PENICILLIN

TO THE EDITOR:—*I am impressed by the article "Eight Fatal Anaphylactic Reactions to Penicillin" by Dr. Abraham Rosenthal* (New York J. Med. **54**:*1485 [May 15] 1954). *Facing the threat of penicillin anaphylaxis, it seems to me that the general practitioner will have to revise his antibiotic therapy. I would appreciate an up-to-date outline of antibiotic therapy for the general practitioner, who must make his decision at his office or at the bedside without sensitivity test of causative organisms. Should penicillin therapy, orally and parenterally, be abandoned? Should the antisyphilitic treatment with 9 million units in the average case be changed?* *M.D., New York.*

ANSWER.—In the past few years there has been an increase in the number of fatal reactions following the administration of penicillin. The incidence of anaphylactic-like reactions is still extremely low when one considers its wide use. Over 300 tons of penicillin were used domestically in 1953. A complete revision of the general practitioner's use of antibiotic drugs is not warranted on the basis of the above report, but patients should be questioned concerning previous reactions to penicillin. If any reactions have occurred, the use of penicillin should be carefully weighed, keeping in mind that, for many infections, other antibiotics are available. Patients who have a previous history of allergy to foods, drugs, bacteria, or pollens may be skin-tested intradermally if it is decided that penicillin is the drug of choice. Such tests are not infallible, but the patients who are extremely sensitive to penicillin are the most likely to show a positive wheal at the site of the intradermal test. If it is decided to use penicillin in spite of past allergic history, the physician must have epinephrine and antihistaminics available for immediate injection if necessary.

Penicillin is still the drug of choice in many infections, and its use should not be abandoned. The oral administration of this drug is less apt to produce fatal anaphylactic reaction than its parenteral use. Accordingly, at the bedside, where skin tests may not be practical, tablets and suspensions of penicillin may be substituted for injections. Since anaphylactic reaction in some patients appears to be related to inadvertent intravenous administration, the physician should never fail to attempt aspiration before injection of penicillin to make certain that the lumen of the needle is not within a blood vessel. Penicillin is the drug of choice in the treatment of syphilis, and dosage schedules for various stages of this disease are given by Curtis and others (*J. A. M. A.* **145**:1223 [April 21] 1951).

XANTHOPIA FOLLOWING SNAKE BITE

TO THE EDITOR:—*Have you any record of a snake-bite victim complaining of yellow vision?*

S. B. Forbes, M.D., Tampa, Fla.

ANSWER.—In xanthopia the color perception is abnormal, so that a patient sees everything as if through yellow glasses. The classical example of xanthopia occurs after ingesting santonin. Other drugs, such as amyl nitrite, picric acid, chromic acid, and large doses of digitalis may produce colored vision. More recently abnormal color perception has been reported after use of streptomycin. Many kinds of pathological ocular function, including bloody injection of the ocular apparatus, disturbances of accommodation, amblyopia, amaurosis, and even total blindness, have occurred following snake bite. Ptosis or paralysis of the eyelids is quite characteristic after cobra bites. The great reference books on toxicology, such as those of Kobert, Lewin, and Blyth, do not mention xanthopia or any other color disturbances of vision, but a book by Paresh Banerji entitled "Handbook of Snake-bite" (London, Butterworth & Co., Ltd., 1929) speaks on page 38 of a rare perversion of sight in which a victim after snake bites "sees everything in red." Nevertheless, in the histories of 1,134 cases of poisoning by bites of all kinds of snakes in this handbook no xanthopia is reported, although other disturbances of the eye and its appendages are very frequently mentioned. The only other work referring specifically to xanthopia is by Hilbert, who published an article entitled "Gelbsehn nach Schlangenbissen" (*Memorabilia* [new series] **18**:1-15, 1900). It deals with ocular disease after snake poisoning. Now that certain snake venoms are used therapeutically, it is interesting to note that cobra venom in experimental psychological tests actually improved the acuity of vision and enlarged the field of vision in 35 normal persons (Macht and Macht: *J. Exper. Psych.* **25**:481-493, 1941).

ORANGE JUICE, MEAT, AND MILK

TO THE EDITOR:—*How much orange juice will provide the alkalinizing effect of 1 gm. of sodium or potassium citrate? How much available potassium is provided by 50 cc. of orange juice? What percentage of uncooked muscle meat is available as net protein to the consumer? How much cow's milk gives 1 gm. of calcium to the imbiber?* *M.D., Pennsylvania.*

ANSWER.—H. C. Sherman's book, "Chemistry of Foods and Nutrition," states that the alkaline effect of 100 gm. of orange juice is about equivalent to 5 ml. of normal alkali. Therefore, 1 gm. of anhydrous sodium citrate would be roughly equivalent in alkalinizing effect to about 230 ml. of average orange juice and 1 gm. of anhydrous potassium citrate would be equivalent to about 195 ml. of juice. According to Bills, McDonald, Niedermeier, and Schwartz (*J. Am. Dietet. A.* **25**:304, 1949) the potassium content of orange juice is about 190 mg. per 100 gm. Therefore, 50 ml. of orange juice would contain about 95 mg. of potassium. Whether all of this is in an available form is not known. Uncooked lean beef contains 18 to 20% of protein. After cooking the protein content varies from about 25 to 27% because of the decrease in moisture. Average whole milk contains 118 mg. of calcium per 100 ml., according to U. S. Department of Agriculture Handbook No. 8, "Composition of Foods, Raw, Processed, Prepared." In order to obtain 1 gm. of calcium it would be necessary to ingest about 850 ml.

SHOWERS AND APOPLEXY

TO THE EDITOR:—*Is there any evidence that habitual cephalic warm and cold showers started early in life help to maintain cortical and subcortical vascular elasticity, thus preventing cerebral apoplxy due to arteriosclerosis?*

C. D. J. Generales, M.D., New York.

ANSWER.—So far as it has been possible to ascertain, there are no evidences of either beneficial or detrimental effects of warm and cold showers on the pathogenesis of cerebral arteriosclerosis, and there are no physiological reasons why this procedure should have any appreciable effect on the probability of apoplexy. The prevention of apoplexy is essentially the prevention of cerebral arteriosclerosis. External heat or cold do induce changes in the peripheral arterial tension (as illustrated by the cold pressure test of Hines and Brown in early or potential hypertensive disease) and thus by changing the intravascular pressure in the cerebral vessels affect the intracranial circulation. There is much reason to doubt whether intracranial arteries and/or arterioles have the capacity to expand or contract to any significant degree. In view of the impression that apoplexy due to thrombosis in narrowed sclerotic vessels may be precipitated by relative hypotension (sleep, anesthesia, or perhaps, external heat) and that the trigger setting off the cerebral hemorrhage through the weakened sclerotic arterial walls may be an

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The answers here published have been prepared by competent authorities. They do not, however, represent the opinions of any official bodies unless specifically so stated in the reply. Anonymous communications and queries on postal cards cannot be answered. Every letter must contain the writer's name and address, but these will be omitted on request.

acute rise in the systemic arterial pressure (rage, fear, violent effort, cold plunge, etc.) it could be argued that warm and cold showers might predispose to apoplexy rather than prevent it.

ERYTHROMELALGIA

TO THE EDITOR:—*I saw a 5-month-old boy who for a month had been having attacks of redness of the feet with obvious local heat in the feet and pain during the attacks. The episodes last for 20 to 30 minutes and are precipitated by holding the baby with the feet dependent and probably by application of heat. The attacks appear to fit the description of erythromelalgia. What other disease should be considered in the differential diagnosis? What is the treatment and prognosis?*

J. H. Rogers, M.D., Gadsden, Ala.

ANSWER.—From the description, the findings are consistent with the diagnosis of erythromelalgia. While this disease has been observed at every age, it is rare below the age of one year. Erythredema polyneuropathy must be differentiated from this disorder in infants. It presents red and painful extremities and is commoner at this age period. The clinical findings in erythredema polyneuropathy are, however, quite distinctive and dramatic and differ from those of erythromelalgia in that, in addition to the local findings, the child is alternately irritable and restless and then apathetic. This disease is persistent rather than episodic, and the hands and face are prominently involved. There is edema, desquamation, and profuse sweating. Photophobia is marked in most patients, and alopecia and salivation are generally present. If such a clinical picture is present, erythredema polyneuropathy must be considered and appropriate studies should be undertaken, such as examination of the urine for arsenic or mercury. Erythromelalgia, on the other hand, presents no such clinical findings. The most prominent symptoms are pain and hyperemia, which are usually localized in the feet and are especially marked if the feet are dependent or their temperature is increased. The symptoms appear at intervals at first; later they may be persistent. Erythromelalgia in some patients accompanies organic vascular diseases, while in others it appears to be due to stimulation of centripetal vasodilator fibers of the posterior roots or peripheral nerves. Syringomyelia and cord tumor have been found in some patients. In most of the patients studied, however, no organic disease has been found and hyperirritability of the vasomotor mechanism occurring in neuropathic patients has been suggested. Little or no information is available on the prognosis of erythromelalgia in infants. Trophic changes generally ensue in the involved feet, and hyperesthesia becomes marked. The treatment in the absence of a specific cause is symptomatic. The affected extremities should be kept horizontal to diminish the vasodilatation and associated pain. Cool baths often afford relief of pain, but epidural injections of local anesthetics have not been tried on enough patients to warrant any conclusions as to their efficacy.

TREATMENT OF NEURITIS DUE TO ANEMIA

TO THE EDITOR:—*What is the latest treatment for neuritis following pernicious anemia?*

Joseph A. Oliver, M.D., Rockwell, N. C.

ANSWER.—Cyanocobalamin (vitamin B_{12}) and liver extract constitute the only effective treatment for the neurological manifestations of combined system disease associated with pernicious anemia. There is no evidence that other measures are of any value. It is probably worthwhile to give as much as 1,000 mcg. of cyanocobalamin intramuscularly once or twice a week for five or six injections in order to make certain that the tissues contain optimal amounts of the vitamin, but there is no convincing evidence that these large doses are therapeutically superior to injections of 30 to 50 mcg. once or twice weekly. As long as cyanocobalamin is given in these amounts, there is no contraindication to the simultaneous administration of folic acid, but the latter is of no value in the treatment of the neurological changes and is not required for effective treatment of the anemia. If folic acid is given without adequate amounts of cyanocobalamin, the neurological symptoms may become more severe.

MUMPS VACCINE

TO THE EDITOR:—*What is the efficacy of mumps vaccine? Can it be combined in the same syringe with diphtheria-tetanus toxoid? In the past school year 380 cases of mumps occurred in 2,200 Navaho students. We anticipate about 500 new students 10 years old or older next fall. The new students have had no previous schooling and hence supposedly very little exposure to mumps. Should this new group be vaccinated against mumps? What is the duration of the resulting immunity? Would repeat vaccinations be required?*

M.D., Utah.

ANSWER.—Although reports on the clinical efficiency of mumps vaccine are meager, some physicians who have used it are favorably impressed with its value. The combination of mumps vaccine in the same syringe with diphtheria-tetanus toxoid might be ill advised. Ordinarily diphtheria-tetanus immunization would be undertaken before one year of age—generally at about 3 months. Mumps is rare during the first 12 months of life. Prior to puberty mumps complications, with the exception of encephalitis, are also rare. If children have been exposed it is probably better for them to have mumps than to face the possibility of acquiring the disease later during adult life. If there have been no active immunizations in students who are 10 years old or older there are likely to be some fairly severe reactions when diphtheria-tetanus toxoid is administered. Mumps vaccine is grown in chick embryos. Therefore, anyone allergic to egg might experience a severe reaction that could intensify any sensitivity due to the diphtheria-tetanus toxoid. For statistical purposes and clinical observations, it would be interesting to vaccinate one-half of the new pupils. Immunity obtained by mumps vaccine lasts 6 to 12 months. To control an outbreak of mumps, vaccine would probably have to be repeated every six months.

TENDERNESS IN FOOT

TO THE EDITOR:—*What is the cause and treatment of a subacute tenderness to finger pressure and hand squeezing along the lateral side of the left foot only? The patient is 50 years old and has no signs of shoe pressure and no other symptoms. The tenderness does not involve the heel or toe, only the midportion of side and adjacent dorsum.*

Wm. S. Levy, M.D., Woonsocket, R. I.

ANSWER.—Without any other findings or a history of trauma this case would probably be the result of foot strain. Since the outer side is the strong part of the foot such a strain would presuppose the wearing of a stiff-shanked shoe or some other type of arch support. Strain of the lateral side of the foot is difficult to relieve with supportive measures. Treatment will require a decrease in the amount of time the patient stands or strains his foot. Relief of the strained tissue may be obtained by rest, hot moist packs, and light massage. Simple adhesive strapping with the heel in varus will permit function while recovery occurs.

PULSATION OF BRACHIAL ARTERIES

TO THE EDITOR:—*Is there any significance to the sign of visible pulsation of the brachial arteries?*

Joseph V. Waitkunas, M.D., Woodhull, Ill.

ANSWER.—In the younger age group, visible pulsations of brachial arteries are sometimes seen in thin persons or when the arteries are unusually close to the surface, and such is of no clinical significance. In middle-aged persons, visible pulsations are not unusual, especially when there has been muscle wasting. Such pulsations alone are of little significance. However, if the arteries on palpation are hard and tortuous, it is a definite sign of arteriosclerosis. Also, aneurysm of the brachial artery will cause visible pulsation, but this is unilateral. Visible pulsations of the brachial arteries should be considered in connection with the examination of the other arteries, such as the radial and the retinal vessels.

SEDIMENTATION RATE AND ANTICOAGULANTS

To the Editor:—*What changes in the sedimentation rate occur before, during, and after anticoagulant therapy such as is currently used in treating myocardial infarction?*

G. E. Huston, M.D., Marietta, Ohio.

Answer.—Early in the era of anticoagulant therapy, it was reported that the sedimentation rate was increased by the use of anticoagulants. A careful investigation made by Wright and Prandoni (*J. A. M. A.* **120:**1015 [Nov. 28] 1942) on the effect of bishydroxycoumarin (Dicumarol) on this phenomenon has established that this is actually not the case. Later studies by the same workers and others confirmed these findings and established that the same was true for most of the anticoagulants in clinical use today. Increase of the sedimentation rate is rather commonly associated with the diseases for which anticoagulants are given, such as myocardial infarction, thrombophlebitis, and pulmonary embolism, but, even though anticoagulant therapy is continued, if the disease process in a patient subsides and there is no other cause for an increase in the sedimentation rate, it will return to normal levels.

X-RAY THERAPY

To the Editor:—*Are there any long-range adverse effects from the therapeutic use of x-rays in the treatment of osteoarthritis of the lower back? This request is predicated on the presumption that the diagnosis is correct and the therapy is being rendered by a competent radiologist.*

A. Morris Ross, M.D., New York.

Answer.—If the assumption can be made that the x-ray therapy in the case of osteoarthritis of the lower back is given by competent radiologists, there should be no risk whatsoever of delayed or long-range adverse effects. The doses of x-rays usually used lie well within the limits of safety.

PREGNANCY TEST

To the Editor:—*Please send me details of the technique used in running the pregnancy test on Rana pipiens.*

M.D., Alabama.

Answer.—The details of the technique used in performing the pregnancy tests on Rana pipiens can be found in either *Endocrinology* (**43:**349, 1948) or *Science* (**107:**198, 1948). In brief the procedure is as follows: 1. The first morning urine specimen is obtained. 2. Five cubic centimeters is injected subcutaneously in the dorsal or lateral lymph sac of the male frog; two or more frogs are used in each test. 3. The injected frogs are set aside in glass jars for four hours. 4. Urine is obtained from the cloaca with a fine pipette at two and four hours. 5. The urine is examined microscopically, and the presence of sperm in the urine indicates a positive test.

BCG VACCINE

To the Editor:—*In* The Journal, *July 3, 1954, page 949, is a query on BCG vaccine. The reply contains a statement to the effect that this vaccine would do no particular harm. Such a statement has long been challenged by many physicians. I believe the readers should know some of the reasons for this challenge.*

When Calmette and Guérin gave this bacillus their names in 1921 and declared it a virus fixé in 1924, it was so reduced in virulence that it would not produce tubercles in animal tissues. However, since that time significant changes have occurred in the cultures. Suter, Schaefer, and Dubos (Proc. Soc. Am. Bact., *1951, page 90) have carefully examined BCG cultures from several laboratories, including the two mentioned in this reply. They found no two alike even on gross culture appearance, and they found that each culture examined was composed of multiple bacterial forms instead of the single bacterial form in the BCG produced by Calmette. Moreover, Pierce, Dubos, and Suter* (Proc. Soc. Am. Bact., *1951, page 90) observed that among these multiple forms there was considerable variation in the invasiveness of animal tissues. This leaves no doubt that BCG cultures have undergone marked changes since Calmette designated his original one a virus fixé. Apparently no two cultures examined have undergone the same changes. These changes probably account for the alarming occurrences following administration of BCG to persons and animals during the last 25 years. In so many persons who received BCG subcutaneously, abscesses and ulcers developed at the sites of administration and disease of regional lymph nodes broke down and discharged pus for months, so that scarification and puncture methods of administration were recommended. Although this reduced the incidence, there are still many such cases reported each year. These are clinical lesions that behave like those from natural infections. Some of them require treatment including surgery and antimicrobial drugs. Dubos* (Am. Rev. Tuberc. **60:***670, 1949) found that, when BCG was introduced into mice on deficient diets, progressive and sometimes fatal pulmonary tuberculosis developed. Vorwald, Dworski, and Pratt* (Am. Rev. Tuberc. **62:***455, 1950) produced progressive and killing tuberculosis in silicotic guinea pigs with BCG from two different laboratories. Hauduroy and Rosset* (Compt. rend. Acad. d. sc. **232:***445, 1951) produced progressive tuberculosis in normal golden hamsters and ground squirrels. When BCG was introduced intraperitoneally, all golden hamsters were dead from widely disseminated tuberculosis in four months. When introduced intracutaneously, all animals were dead in 12 months.*

Many persons who have received BCG have later had clinical tuberculosis, and a considerable number have died from this disease. Attempts to determine the type of bacilli causing illness and death have been far too few. In The Journal, *May 1, 1954, page 61, attention is called to seven cases of lupus vulgaris having developed from BCG. This was prophesied 12 years ago by J. A. Jensen of Denmark, because he found the virulence of BCG was only a little less than that of the usual tubercle bacillus. In the June 19, 1954, issue of* The Journal, *page 773, a well-documented case of tuberculosis resulting fatally from BCG is reported. BCG was administered when the man was 20 years old. This resulted in a reaction to tuberculin within six weeks. The first clinical lesion appeared about a year later. From then on numerous clinical lesions developed in various parts of the body including the lungs and kidneys. He died from tuberculosis in December, 1953, and BCG was recovered from the numerous lesions. Such cases make one strongly suspect that in the past some clinical and some fatal cases of tuberculosis in those who previously received BCG were due to the BCG itself rather than its failure to protect against natural infection. In any event, the well-documented evidence regarding BCG cultures and the disease it produces in animals and persons is cause for alarm and justifies challenging the statement in this reply, or wherever it is made, that BCG does no particular harm.*

J. A. Myers, M.D.
111 Millard Hall
University of Minnesota
Minneapolis 14.

To the Editor:—*I am concerned regarding the answer in the July 3, 1954, issue of* The Journal, *page 949, to a question on BCG vaccine. I feel that the reader would have the impression that this so-called vaccine could do no harm. In recent years an impressive amount of information has been gathered in studies on cultures, animals, and human beings that forces one to challenge a statement to the effect that BCG is without harm. After approximately 40 years of use, BCG remains a controversial question in all parts of the world. Most certainly great caution is justified.*

Seymour M. Farber, M.D.
University of California Tuberculosis Service
San Francisco Hospital
22nd St. and Potrero Ave., San Francisco.